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THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

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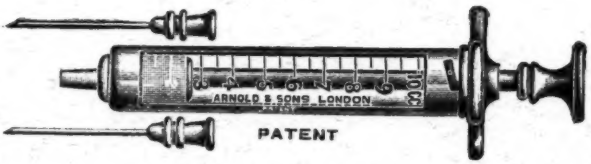

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THE

VETERINARY JOURNAL

NOVEMBER, 1910.

Editorials.

THE PAYMENT OF MEMBERS OF THE COUNCIL OF THE ROYAL COLLEGE OF VETERINARY SURGEONS.

THE discussion at present being carried on in political circles on the question of the payment of Members of Parliament has its counterpart in a very small way in the politics of our own profession. The members of the Council of the Royal College of Veterinary Surgeons have always given their services freely, and we believe ungrudgingly, in the interests of the profession to which they belong. The profession as a whole is indeed grateful to those gentlemen, and they have need to be. Those gentlemen have not only given their time and services, but they have also paid their own expenses, which in the case of members from a distance must be very considerable. This is all very well in its way, but it has had one effect which in the eyes of some members of the profession is not quite desirable. It has resulted in a majority of the councillors hailing from the South, and from large towns so that country practitioners, and those in districts remote from London consider that they are not adequately or even directly represented on the ruling body. It is useless to retort that they have the vote and so can take part in the elections, but how can a small body of men such as, say, the Welsh practitioners, or those in the North of Scotland hope to outvote the numerous London and suburban practitioners? We are afraid they cannot hope to do so. There is, however, another

very important cause contributing to the present state of affairs, viz.—the difficulty of getting suitable men from long distances to consent to be nominated for the Council owing to the great expense entailed. The late Mr. W. Byrne repeatedly stated to us that his actual out-of-pocket expenses during his term on the Council were over £50 per annum, to say nothing of the loss he must have sustained by the neglect of his practice while attending meetings in London every quarter.

A few weeks ago a country practitioner from the North was complaining to us about some fancied grievance he had against the Council. He said the Council did not care a rap for the country practitioners, as they themselves were almost all town practitioners or elderly men who had "feathered their nests." We did not agree with the statement, and when he was taken to task and asked to mention any other good business man, a country practitioner, who would be prepared and feel justified in neglecting his practice, and at the same time willing to pay anything approaching even £25 a year out of his own pocket for the benefit of his profession, he was silent. He later confessed that he was one who had protested against being asked to pay one guinea a year for the protection of his own interests, but if there was any probability of the proposed new Bill remedying this state of affairs he would withdraw his opposition and gladly contribute an annual fee.

We do not suggest for one moment that members of Council should be paid for their services, and we do not believe that any one of them desires anything of the kind. To be elected to the Council is about the only honour our profession can bestow upon its members, and recognizing that, the councillors give their services freely and ungrudgingly, and do the best they can with the funds at their disposal for the general welfare of the profession.

But ought we to ask so much from them? Ought we to put them to such expense as is entailed by their proper attendance at Council meetings? Such a system certainly makes it quite impossible on financial grounds for many good, shrewd, business men to accept the only honour we could confer upon them, and robs us of their valuable services.

Hence it is that we consider one very great reason why the proposed annual fee should be instituted, that members of Council should have their travelling expenses paid. Then it might be possible to get more direct representatives of general practitioners in Wales, Ireland, and the North of Scotland. As at present constituted there is only one general practitioner on the Council from Scotland, and none from Wales or Ireland.

G. H. W.

THE TREATMENT OF ACUTE LAMINITIS.

IN the VETERINARY JOURNAL of last December we recorded an interesting clinical note by Mr. Philp on "The Use of Adrenalin in the Treatment of Acute Laminitis." We added a footnote in support of this line of treatment, and asked for further reports from any practitioners adopting it. It is, consequently, with great pleasure that we record in this issue two cases treated successfully by this method by Captain Jolliffe. Both cases would appear to have been very acute, and that recovery should be so complete in both cases is certainly very gratifying. The injection of adrenalin over the plantar arteries undoubtedly diminishes the amount of blood going to the feet, and in that way diminishes the congestion and pain. If the congestion is early relieved, then as a consequence the probability of exudation between the horny and sensitive laminæ with "dropped sole" and permanent changes is diminished. Hence we consider that this method of treatment, if adopted in the early stages, should reduce the severity of the attack, curtail its duration, and diminish the probability of untoward sequelæ. We should be glad to receive more reports of its trial and the results obtained, whether favourable or otherwise.

G. H. W.

General Articles.

Indexed B. A. I.

THE CAUSATION OF RICKETS.[1]

By FREDERICK W. COUSENS, M.R.C.V.S.

London.

Many and varied have been the causes assigned for rickets. Almost every prejudicial circumstance in the life of the infant—and in that of the parent too, for that matter—has been cited as the etiological factor.

Etiological Theories.

According to Kassowitz and some of the German school, it is of congenital origin, a theory, however, mainly founded, at least by its earliest adherents, on macroscopic appearances alone, and not on the finding of any characteristic histological changes in the bones. In every infant the bones are soft, the abdomen may be protuberant, the fontanelles will be open, and there will be some swelling of the costo-chondral junctions, and yet it was on the ground of these facts alone that such a theory was based. The later advocates of this theory, however, did examine the minute histological changes in the bones, but as much of the material studied was obtained from stillborn children, and as the pathological anatomy of osteochondritis syphilitica and rachitis was at that time only imperfectly understood, these two conditions were much confused, and it is not improbable, therefore, that many of the supposed rachitic lesions were in reality of a syphilitic nature. As these two processes came to be differentiated from one another, each succeeding published record of work done on this subject showed a marked diminution in the proportion of congenital rickets. Kassowitz in 1882 considered that 80 per cent. of all children born were rachitic: in 1897 Tschistowitsch put the proportion at 12 per cent., while more recently (1902) Escher, after investigating a large series of cases both clinically and histologically, was only able in one single instance to make a diagnosis of congenital rickets.

There is, of course, such a condition known and described as "fœtal rickets," but it is considered by the majority of observers an entirely different disease from that which ensues after birth, and is, moreover, as rare as ordinary rickets is common. It will probably be better then, at least for the present, to classify it apart, and designate it by one of its pseudonyms—for example, "osteogenesis imperfecta."

[1] Paper presented at the Meeting of the National Veterinary Association, 1910.

Closely allied to the above theory (congenital origin of rickets) is that mainly advocated by Siegert, who holds that the condition is hereditary, and dependent on some inherited weakness or predisposition. This, after all, is merely a case of begging the question and failing to discuss the main points at issue. Hausen, however, reports the case of a stallion who begot seven rachitic foals. Later, two of the mares, one of which had given birth to three and the other to two of these rachitic foals, became pregnant to another stallion, and bore healthy non-rachitic offspring.

It is during extra-uterine life, however, that most present day authorities believe the etiological factors play their part, and the

FIG. 1.—The two collie puppies, ten days before death. Note the extreme degree of bending of the forelegs in the rachitic animal.

general consensus of opinion would ascribe the disease to incorrect feeding, bad hygiene, and want of sunlight. Of all these possible malign influences it is some error in feeding which receives most credence and support. By some it is said to be due to an entire want of suckling, to the mother not nursing the child long enough, to suckling at too frequent intervals, and by others again to prolonged lactation. Siegert found that out of 845 artificially fed infants 81 per cent. developed rickets, while in 923 infants who had been nourished at the breast for at least four and a half months, only 31½ per cent. became rachitic. On the other hand, Holt mentions that although

negro and Italian children in New York are entirely breast-fed, they are exceedingly prone to develop the disease.

In artificially fed infants and in the feeding of children, almost every possible quality and combination of the different constituents have been credited with the power of inducing the disease. Some say it is consequent on a deficiency of proteid, others on an excess of carbohydrate, but perhaps the majority consider that it is due to a deficiency of fat and proteid with an excess of carbohydrate. This is a combination of factors which, it must be remembered, may be present in the food of both naturally and artificially fed infants. According to Holt and Cheadle it is in the presence of a low per-

FIG. 2.—Longitudinal sections of bones of hind legs from healthy animal showing normal conditions. From rachitic animal showing the thinning of shafts, and great irregularity and widening of epiphyseal lines.

centage of both fat and proteid that rickets is liable to result, while Bland-Sutton teaches us that it is entirely due to a deficiency of fat. The latter's well-known experiments in the London Zoological Gardens with the lion cubs and young monkeys lend great support to his view. By only one change in the general *régime* of these animals, namely, the addition of fat and cod-liver oil to their diet, lion cubs were reared free of rickets for the first time in ten years. Ashby, while lending his support to the idea of a low percentage of fat and proteid, considers that it is not improbable that fermentation

of the carbohydrates may produce toxins which are responsible for at least some of the phenomena of the disease. Heitzman [3] considers that "lactic acid" is the baneful product, and claims to have induced the condition experimentally by its administration. He believes that the lactic acid results from the fermentation of the carbohydrate and irritates the ossifying tissue; but other observers have not been able to confirm this formation of lactic acid, and have failed to bring about rachitic changes by its administration.

Esser [4], who has lately written on the subject, believes that the disease is due to over-feeding, and bases his theory mainly on the similarity of the blood pictures in rickets and chronic over-feeding. He contends that over-feeding produces a chronic gastro-enteritis, and results in a deficient absorption of the food constituents. Cheadle [5], too, in one place speaks of rickets ensuing on starvation, which is, however, contrary to clinical experience.

Considering the fact that the rachitic changes in the bones are in great part characterized by deficient calcification, it is not surprising that some observers have looked for the etiological factor in a defect of lime metabolism. It has been attributed to an absence or deficiency of lime in the food, to a defective anabolism of the lime salts, and, lastly, to an increased katabolism of these same salts—in short, to all the possible variations in the process of calcification. Chossat [2] produced curvature of the bones experimentally by depriving animals of earthy salts, but Friedleben doubted the condition being true rickets. Voit [6] also believes that he was successful in causing rickets by depriving animals of calcium. Katz [7] saw typical rickets in a fowl which had been confined in a narrow cage for some months, and which he considered due to the fact that the bird could not get a sufficient amount of lime. Reimers and Boye [8] also noted some changes in the bones of dogs fed on a diet poor in lime, but very different from that they observed in cases of spontaneous rickets. The bones in the experimental animals were soft, with some thickening of the epiphyses. The bony trabeculæ were merely thinned and did not reveal the presence of any osteoid tissue. In cases of spontaneous rickets, however, the epiphyses were enormously thickened and much osteoid tissue was present. That it is due to a want of lime is unlikely when one considers that farinaceous foods and cow's milk—the diet on which rachitis is supposed to flourish—contain more than an abundance of that substance. There is no doubt, of course, that during the rachitic processes there is a deficient absorption of lime by the newly-formed bony tissue, and just as truly is there an increased

katabolism of the same salts, but it is just on what this perversion of metabolism depends that the crux of the whole question lies.

Hygiene and climate are acknowledged by most authorities to play some part in the etiology of rickets, but only to a slight extent, and in a very general way. To this question, however, I will return later.

The infective theory has been mooted by many, but as yet there is no definite proof in favour of any such idea. Drs. A. Torane and Salvatore Forte [9] believe they have induced the disease in rabbits by inoculating them with watery and alcoholic extracts of the fæces of children suffering from rickets and diarrhœa. Curiously, though rachitis changes are said to follow on injection of either the alcoholic or watery extract, a mixture of the two extracts is quite inactive.

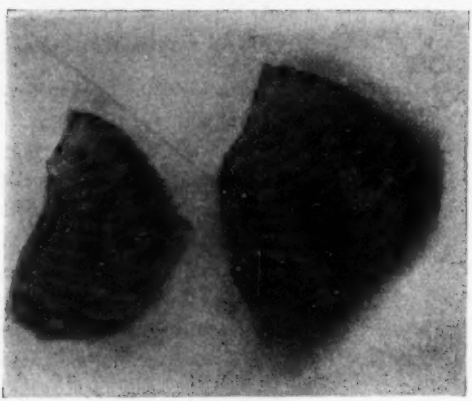

FIG. 3.—Inner aspect of the thoracic walls in healthy and rachitic animals. Rachitic animal showing the marked rosary or nodules at the junction of the ribs and sternum.

Moussu [10] also is of opinion that it is an infectious disease, as he caused a healthy animal to take rickets by confining it in a cage along with a rachitic one; and then, without subsequent disinfection, he confined another healthy animal in the cage, and it also in turn succumbed to the disease. This experimenter, in addition, inoculated various kinds of animals with emulsions of rachitic bone-marrow, but without any effect, unless he injected what he calls a very virulent material, and also confined the animals in the above-mentioned or some other similarly infected cage.

Syphilis, too, has been considered answerable, and it is possible that it may act as a predisposing factor; but that it is the prime etiological cause, as Parrot [1] and Marfan [11] consider, is a view

which has not received much support in this country, and seems, moreover, to be entirely negatived by the fact that rickets can be easily induced in the lower animals.

The Effects of Deprivation of Exercise.

The great diversity of opinion which prevails regarding the etiology of this disease is shown by the above brief account of the various theories, and is sufficient proof that as yet the true etiological factor has not been discovered. Rievel [12] while summing up the recent work on this subject, suggests that rickets may not have the same origin in all cases, and that many factors may play a part in its causa-

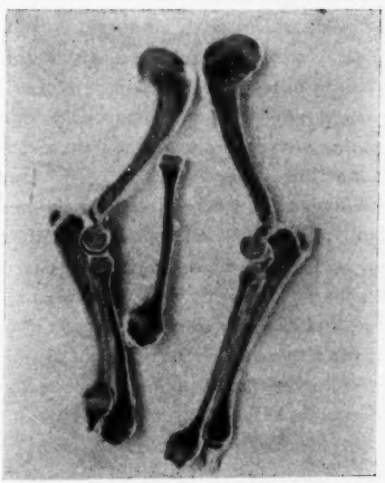

Fig. 4.—Sections of bones of forelegs of the two collie puppies. Healthy puppy. Rachitic puppy: Note the bending of the humerus, and the great thickening of the epiphyseal lines in the radius and ulna of the rachitic puppy.

tion. It is my intention, however, as the result of some experimental work which I have recently carried out, and by a critical survey of the various influences which affect the child, to show that there is one factor, and that a very potent one, in inducing the disease—namely, want of exercise.

Lack of exercise as a factor in the pathology of the disease has received remarkably little attention, though to my mind it is of the utmost importance, and in future will always require to be taken into consideration when discussing experimental rickets. Though confine-

ment is mentioned by several authors, its mode of action is either misunderstood, or it is supposed to work along with some other baneful influence, and to play a subsidiary part. Dudgeon [13] believes that it acts through lack of fresh air and sunlight, while Clement Lucas [14] mentions it as a contributory factor to incorrect feeding, and Esser [4] to overfeeding. By depriving young dogs of exercise I invariably induced the condition, though they received as much fresh air and sunlight as the control animals. Moreover, their kennels were cleaned regularly. It is not an uncommon experience for experimental animals to develop rickets, consequent, I believe, on the confining of the animals in cages, an almost invariable practice in experimental work. In this way we can explain the case of the rachitic fowl reported by Katz, and also the results of Moussu's experiments. It is in a similar fashion that climate exerts its influence. In warm, genial climates the children will be out much during the day, while in temperate and treacherous climates like our own, especially during the winter, children are exceedingly likely to be cooped up in the house for days, and even weeks, at a time.

It was some two years ago, when commencing an investigation of experimental rickets, that my attention was first attracted to this subject. I attempted to produce rickets in puppies by modified feeding-giving them such generally recognized rachitic diets as bread and water, oatmeal and water, and rice and water, and yet not one developed the disease. They invariably wasted, became marasmic, and died, thus confirming the well-known fact that marasmic infants never become rachitic. But the control animals, at least those which did not become atrophic consequent on diarrhœa, though being fed normally—as, for example, on milk and porridge—all became affected.

In the first series of experiments three control animals developed rickets, one of them, however, less severely than the other two. This animal was fed in a similar fashion to the others, and spent most of its time in their company; but, owing to the fact that it was ultimately intended as a companion, it was exercised by its prospective owner once or twice daily. Accordingly it seemed to me not improbable that in the confinement and want of exercise—the only abnormal factor in the *régime* of these animals—one had to deal with the etiological factor. I allowed the comparatively healthy animal to get still more exercise for about a week, when all appearances of rickets practically disappeared. On once more confining him with his brothers and sisters he again rapidly became affected. Two of this litter suffered so severely that they were unable to walk, and merely shuffled about

the floor of the kennel as if they were affected with paraplegia or diplegia. Similar experiments have been carried out on two other sets of animals, with exactly the same result. By subjecting the animals to no other abnormal condition than the want of exercise, marked rachitis has invariably ensued.

The table given comprises my observations in chronological order on sixteen animals. Nine of these were treated by confinement and deprived of exercise, but they were fed normally. One (No. 14) died of marasmus, too early, however, to allow of the development of the disease, but the other eight (Nos. 2, 4, 5, 8, 9, 10, 11, and 15) became rachitic to a marked degree. Two (Nos. 1 and 3) were treated by modified dieting and were allowed exercise. Both died of marasmus, but without showing any signs of rickets, although they lived beyond the age at which the disease usually appeared in the experiments. The other five (Nos. 6, 7, 12, 13, and 16) were used as controls. They were all allowed exercise, but as regards feeding, housing, and hygiene were treated in a similar fashion to those which developed the disease, and they all remained entirely free of any rachitic manifestation.

I will quote in more detail my last series of experiments. I obtained three collic pups, aged 2 months, from one litter. These three animals were kept in one house, were fed in exactly the same manner, on a diet of oatmeal porridge and milk, and were subjected to the same atmospheric conditions as regards amount of sunlight, warmth, and purity of air. Two, however, were confined in a small cage closed on either side but open above, and only covered in front with wire netting, while the other was allowed to run about the room at large and play with a cat. All three at first suffered from diarrhœa, and one of the confined animals in subsequence died, but without showing the slightest evidence of rickets. The other two recovered, and steadily put on flesh, and they increased in weight by almost exactly the same amount each week. The puppy allowed to run about developed along normal lines, while the one confined ultimately became rachitic and presented a very typical picture. Fig. 1, from a photograph, shows the contrast between the two animals. The rachitic pup continued to take his food well, and was not troubled with diarrhœa. The first change noticed was a certain degree of languor. When allowed out of the cage he seldom frolicked about, and he was unable, except with the greatest difficulty, to ascend even a very gradual stair. His legs ultimately became bandy, and the beading of the ribs got more marked. His lethargy increased and he lost strength, and, though almost as large and as heavy as his

sister, he was frequently overturned by her simply knocking against him.

Post Mortem.—The difference between the bones of these two animals was most apparent. In the control the bones were straight and firm, the costo-chondral junctions normal in appearance, and the epiphyseal lines narrow and regular. In the other animal the long bones were curved and exceedingly soft, the epiphyseal lines were broad and irregular (fig. 2), and there was marked swelling with hyperplasia of the costo-chondral junctions.

In this paper I only intend to deal with the etiology of the condi-

FIG. 5.—Photomicrograph of a section of rachitic bone stained with thionin blue as advised by Schmorl for the demonstration of bone cells. Note the presence of much osteoid tissue and the dwarfed processes and irregular arrangement of the bone cells.

tion, and consequently will not devote much space to the pathological changes. In order, however, to show that the condition I had induced experimentally was in reality rickets, it may be advisable to recount the main histological findings in the bones. I contented myself by examining the ribs and long bones. As previously mentioned, the bones were soft and pliable, and in some instances could be flattened between the finger and thumb. This pliancy of the bones was most striking in comparison with the hardness of the bones of normal animals. There was distinct swelling of the epiphyseal ends of the

long bones and of the costo-chondral junctions (fig. 3). Section of the bones revealed enormous thickening of the epiphyseal lines, which measured in some instances 12 mm., and great irregularity and increased vascularity of the same (fig. 4). Microscopic examination showed much hyperplasia and irregularity of the epiphyseal cartilage —deficient calcification at the growing zone with the development of much osteoid tissue (fig. 5). The osteoid tissue was, however, more cellular than that occurring in human rickets, but this can perhaps be accounted for by the more rapid development of the disease in the experiments. The little bony tissue present was composed of cells

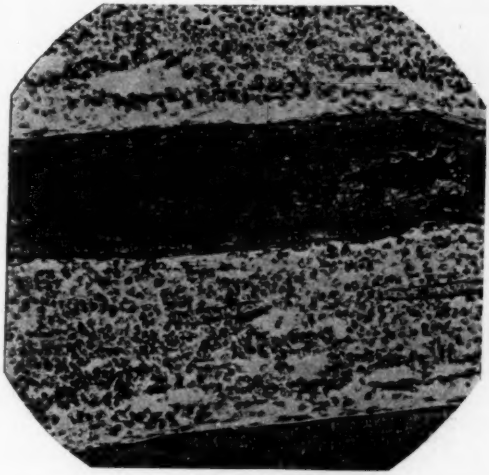

FIG. 6.—Section of ordinary bone for comparison. Here there is no osteoid tissue. The bone cells are regularly arranged, and extend right to the margins of the trabeculæ. In one trabecula some unabsorbed fœtal bone is present.

with dwarfed processes irregularly arranged, and contrasted markedly with what occurs in normal bone (fig. 6).

When we come to correlate these experimental findings with the conditions under which rachitic children live, it will be found that there is much to support this theory of the cause of rickets.

Rickets is a disease of the temperate zone, being very rare, and, in fact, practically unknown in tropical and sub-tropical countries. The staple diet in these climates is anything but nourishing, the people living mainly on rice, or some other similar cereal, exactly the kind of food which is supposed in this country to generate the disease. The

TABLE SHOWING RESULTS OF EXPERIMENTS.

Number	Sex	Date when came under observation	Age when came under observation		Treatment	Date when rickets developed †	Cause of death.	Date of death	Time under observation in weeks
1	M	May 11, 1906	At birth	All of one litter	Dietetic	—	Marasmus ...	Aug. 5, 1906	12
2*	F	,, ,,	,,		Confinement	July 21, 1906	Killed ...	,, 17, ,,	14
3	M	,, ,,	,,		Dietetic	—	Marasmus ...	,, 18, ,,	10
4*	M	,, ,,	,,		Confinement	July 20, 1906	Killed ...	,, 23, ,,	15
5*	M	,, ,,	,,		Modified confinement...	,, 30 ,,	,, ...	,, 28, ,,	16
6	F	April 11, 1907	4 weeks	From one litter	Control not confined ..	—	,, ...	June 18, 1907	10
7	M	,, ,,	,,		,, ,, ...	—	,, ...	,, 17, ,,	10
8*	F	,, ,,	5 weeks	From one litter	Confinement	May 14, 1907	,, ...	,, 9, ,,	8½
9*	F	,, ,,	,,		,, ...	,, ,,	,, ...	,, 13, ,,	9
10*	F	,, ,,	,,		,, ...	,, ,,	Broncho-pneumonia	,, 21, ,,	10
11*	M	,, ,,	4½ weeks		,, ...	,, 12, ,,	,,	,, 9, ,,	8½
12	F	June 3, 1907	8 ,,		Control not confined ...	—	Killed ...	July 7, ,,	4½
13	F	,, ,,	9 ,,		,, ,, ...	—	,, ...	,, ,,	4½
14	M	Nov. 30, 1907	8 weeks	From one litter	Confinement	—	Marasmus ...	Dec. 31, ,,	4½
15*	M	,, ,,	,,		,, ...	Jan. 7, 1908	Killed ...	Feb. 4, 1908	9
16	F	,, ,,	,,		Control not confined ...	—	,, ...	,, ,,	9

* Animals which became rachitic.

† These dates are of course only approximate, it being impossible to say exactly from clinical appearances alone when the disease commenced, but at these dates the animals were undoubtedly rachitic.

mothers in these countries no doubt live under more natural conditions and suckle their infants; and, as is well known, in Japan, where rickets is extremely rare, and also in Italy, the mothers suckle their children for unduly long periods, and yet with no untoward results. The climate permits these races to spend much of their time in the open air, and the houses are merely used as sleeping apartments. It is this living so much in the open air which probably accounts for their immunity. As previously mentioned, negro and Italian children, though not subject to the disease when reared in their native lands, become almost invariably affected when dwellers in a city, as in New York, where they are said to be the worst-housed people in the States. The fact that rickets occurs chiefly during the winter months lends further support to this theory. It is during the spring that the city dispensaries are crowded with rachitic children, as a result of the winter's confinement. During the winter, in such a climate as ours, a mother, in order to do her child justice, must be on the watch for, and take full advantage of, every dry half-hour during the day—rather an arduous task for even the best disposed of mothers.

It is a disease which is found chiefly among the children of urban populations, being comparatively rare in the country. Further, it is among the children of the poor and the working classes that we find the disease most frequently, and not among those of the rich. One of the main differences between these two classes of children—the rich and the poor—is that the former has usually a nurse to himself, while the latter has to be content with as much or as little spare time as his almost invariably overwrought mother can devote to him. The one is taken out into the fresh air several hours daily, and is entertained at home in an airy nursery, while the other is induced to lie quiet in bed or in some corner of the kitchen, and give as little trouble as possible. The poor man's child undoubtedly gets out little; his mother may have too many children and has no spare time, or she may not perhaps have the inclination to take her child out for an airing. Or it may be that he is the child of some single or widowed woman, who, in order to earn her livelihood, puts him out to a day-nursery, where he may be fed and cleaned regularly, but of exercise and entertainment he will receive a minimum. Moreover, the uneducated are notoriously afraid of fresh air in case their children may catch cold by exposure to the weather, so that, with good intention however, the child may not be out for days or weeks at a time, more especially should he be so unfortunate as to suffer from some bronchial catarrh. Again, in cities the poor live in tenements up many flights of stairs, so that there is

every inducement for the mother to stay indoors. Well-to-do people find living in tenements a deterrent to going out—how much more, then, must this mode of living affect the irresponsible poor. When the labouring man's child is taken out for a walk he is almost invariably tightly bandaged to his mother's side with a large shawl, which must impede the movements of his limbs and deprive him of almost all exercise. The great variation in the incidence of this disease among the poor in the country and in the town may, perhaps, be accounted for by the very different conditions under which they live. The country poor live in cottages, so that the children can be taken out of doors with a minimum of trouble, the very opposite to that which prevails in the town.

FIG. 7.—Rachitic Great Dane three months puppy. (Skeletons on following page.)

This confinement of the children, which we find wherever rickets prevails, does of course deprive them of some fresh air and sunlight, and thus reduce their resisting powers, but it is not entirely, or even mainly, on these grounds that it exerts its baneful influence, as my experiments prove. It is due to the want of exercise which invariably goes along with, or is consequent on, the confinement. Children confined to the house are much more lethargic than those taken out of doors. They sleep more, are less vigorous, and are content to lie at rest without evincing any desire for the exercise of their limbs. Every observant parent and physician must have noticed this, and in the

case of a healthy child it is possible from this fact alone to say whether or not he has been out for his usual airing.

As regards feeding, much of the evidence is of an exceedingly doubtful nature. I cannot believe that the poor in the town feed their children so much less intelligently than those in the country as to

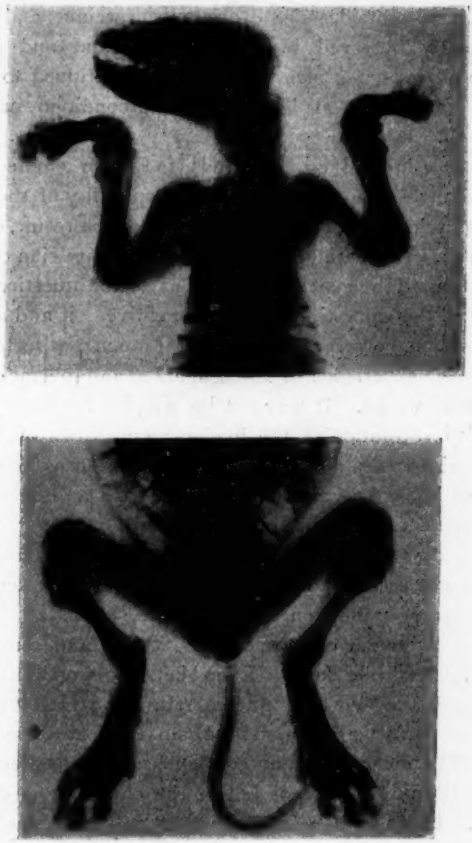

FIG. 8.—Radiographs of Skeleton showing Rickets. Great Dane pup, three months.

account for the great preponderance of the disease among their offspring. Their children are often overfed, but is that not equally true of the well-to-do town child? Does the rich parent never overfeed his child, and does the well-to-do mother invariably, or even usually,

suckle her infant? As is well known, several observers have been unable to induce rickets experimentally by incorrect feeding. Further, is it not during the summer months, when gastro-enteritis is rife and there is consequently a diminished absorption of food, that rickets improves, while in the winter time, with no gastro-enteritis, rickets flourishes? It is questionable, too, if a child, deprived of a sufficient supply of fat and proteid, will steadily put on weight, and yet such is not infrequently observed in both experimental and spontaneous rickets. In all my experiments the animals continued to increase in weight, for some time at least, after the condition had definitely declared itself.

Rickets is admitted on all hands to be an eminently curable disease, its dangers depending on the susceptibility of the children to pulmonary mischief, and on the deformities which result, especially in the female sex. In treating the condition, the physician often pays as much attention to the general hygiene as to the question of feeding, and he may at the same time prescribe cod-liver oil and a hæmatinic, of either of which, however, it is doubtful if much is absorbed. Everyone has had the experience of seeing marked improvement take place in two or three weeks. It may not be possible for these poor people completely to revolutionize the hygienic conditions of their homes, but it is certainly possible to take the child out more often in the open air, to give him a daily bath, and to practise a little massage and passive movement of the limbs. Much more easy is it, to my mind, materially to alter the hygiene of the child than the manner of feeding, even to a slight extent. Will a child who has been accustomed for many months to "the run of the house," with all its attendant delicacies, allow much curtailment of its diet, and remain quiet on a simpler and healthier fare? Even should this revolution in feeding take place, will the intestinal mucosa return so speedily to its normal, and allow of perfect absorption? Again, there are cases in which it is impossible to detect any fault in the manner of feeding, though the mother will frankly admit not taking the child out in case it might catch cold. Holt, though he holds that rickets is due to incorrect feeding, admits that he cannot find any error in the dieting of the negro and Italian children in New York.

In the majority of cases a child develops rickets before it has learned to walk, that is, when it is still dependent on its mother and nurse for its exercise. There are many cases, however, in which the child has commenced to walk only to go off its feet later and become markedly rachitic. Perhaps in most of these instances the condition

is secondary to some illness, as, for example, measles with bronchitis; and, in order to avoid a recurrence of the complication, the child has been kept indoors, and guarded against the slightest draught of fresh air. But there are other cases in which there is no such history of an intercurrent illness, but owing to the mother being pregnant, or ill from some other cause, the child has not been taken out sufficiently often. In neither of these classes of cases can we call to our aid as a cause the manner of feeding: for months the feeding has been the same, and in many instances such as we cannot take the slightest exception to. In all, however, we get the same story of confinement, and the explanation of the condition lies in the want of exercise.

It is on the ground of the above experiments and facts in the child's history that I am forced to the conclusion that want of exercise is the chief etiological factor in this unfortunately too common malady. It is possible that there may be a toxin responsible for the immediate results, but without lack of exercise this toxin will not produce any injurious effects. We may surmise that the lack of exercise and fresh air may be productive of a certain amount of perversion of metabolism, which allows of the generation of some harmful product, and so by auto-intoxication brings about disease. But until this specific toxin is isolated and its nature and mode of formation understood, any such idea is mere theorizing and an easy refuge for ignorance. Typhus fever does not arise *de novo*, and yet we know that somehow or other overcrowding allows the disease to proclaim itself, and by legislation against overcrowding this disease has been kept at bay. Overcrowding and typhus fever are no more closely related to one another than lack of exercise and rickets, and by instilling this fact into the minds of mothers, and especially those of the poorer classes, as well as into corporations who have the welfare of the infant race at heart, rickets would undoubtedly become a very rare disease. Far be it from my intention to belittle dietetics, which is one of the most important branches of pediatrics. Faulty feeding is the cause of much of the infantile mortality, but that it plays any important part in the etiology of rickets is very doubtful.

Conclusions.

(1) Not one of the many theories which have been elaborated to explain the cause of rickets has been universally accepted, and they all lack, not only from the clinical but also from the experimental aspect, unequivocal proof.

(2) It is some error in feeding which, in this country and America,

is commonly believed to bring about the disease, but it is doubtful, however, if feeding plays any important part in the etiology of rickets. Experimentally I, like several other observers, have been unable to cause the condition by improper feeding.

(3) By confining young dogs and depriving them of exercise, rickets has been invariably induced, as in the experiments detailed, and that although their diet was beyond suspicion, the air which they breathed pure, and their kennels were kept scrupulously clean, whereas control animals allowed exercise, but otherwise similarly treated, did not become affected.

(4) Examination of the conditions under which rachitic children are reared reveals one constant and invariable factor in their lives, namely, confinement. Alike, then, on clinical and experimental grounds I accordingly conclude that confinement, with consequent lack of exercise, is the main factor in causing the disease.

[We are indebted to the N.V.A. for the loan of blocks illustrating this article.]

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GANGRENOUS MAMMITIS OF SHEEP.

(A MILD OUTBREAK ASSOCIATED WITH A MICROCOCCUS EXTREMELY VIRULENT IN THE PURE STATE.)

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ALTHOUGH comparatively rare in New Zealand, this disease has on previous occasions come under the notice of the Department, one outbreak having been reported in 1903. The causal organism is a micrococcus originally discovered by Nocard, in France, where the disease is known as "udder disease," *araignée*, &c. The disease is also known in Great Britain under the popular designation "black udder," from the characteristic appearance of the affected vessel.

Description.

The following is a short description of the disease as generally observed: Death usually occurs in two to five days after illness is noticed, although cases of longer duration may occur. The udder is hard, hot, painful, and of a dark-red colour when first seen. Rapidly an œdematous condition spreads along the skin of the floor of the abdomen, and often down the thighs. The skin covering the udder is erysipelatous in appearance; it becomes darker and almost black in colour, and finally gangrene of portions sets in, the skin being readily detached over the portions of cold dead tissue. The milk is scanty, curdly, and often blood-tinged. The temperature is generally high. Emaciation and prostration proceed rapidly, and even very soon after the local lesion is observed general weakness is apparent.

In Europe, Nocard and others state the disease is probably frequently conveyed by the hand of the milker, but obviously that cannot be the medium here. Although Nocard failed to transmit the contagium by painting the teats of udders of healthy sheep with virulent cultures, nevertheless he considered the ordinary means of entrance is by the teat-duct.

Post-mortem Appearances.

The *post-mortem* appearances are as follows: The skin and subcutaneous tissue are infiltrated with a red-tinted œdematous fluid, and sometimes with gas-bubbles. The udder is dark-violet in colour, and infiltrated by a reddish serosity which distends the interstitial tissue. The milk contained in the reservoir and the large canal is coagulated. The inguinal (mammary lymphatic) glands are enlarged, infiltrated, and greyish-red on section. The viscera are engorged, the spleen is small and dark, the heart-muscle is often friable and pale. The muscles are pale, and infiltrated with small hæmorrhages. Microscopical examination of the udder shows the micrococci as a thick mass on the walls of the ducts and sinuses of the udder.

Cause.

The organism is a small diplococcus, found in great numbers in the milk. It stains by Gram's method, and grows readily in all media, with or without air. It liquefies gelatine, and curdles milk rapidly, rendering it strongly acid.

Experimentally, the goat, when the substance of the udder is inoculated, only develops a hard painful tumour, which disappears at the end of ten to fifteen days.

The horse, calf, pig, dog, cat, fowl, and guinea-pig are refractory to large doses. The rabbit develops a hot, painful swelling at the seat of inoculation, which ultimately becomes an abscess, the pus being full of cocci.

The ewe in milk can be readily infected with a small quantity of culture, or milk from an acute case introduced into the teat-canal, the result of which is rapid evolution of all the symptoms above described.

Such are the different features of the disease, as described by Nocard and others, and as we have found, both naturally and experimentally, in New Zealand. In the outbreak to be described, however, the natural disease was not so acute, although the specific coccus itself possessed greater virulence for most animals when inoculated in pure condition than has been recorded by other observers.

Symptoms.

Five pure-bred ewes on a Canterbury property were seen by Mr. Reakes and Mr. Neale, who found a condition of acute mastitis, with rise of temperature, disinclination to feed, &c. The udder was tense and somewhat swollen, and the milk was curdled, but there was no marked evidence of discoloration of the skin, or of gangrene or subcutaneous œdema. The animals had been affected for some days, but there was no typical symptom of gangrenous mammitis. Pipettes of udder secretion brought to the laboratory showed, on microscopical examination, numerous leucocytes, many of which contained diplococci, free diplococci and a large number of very small bacilli, which stained but comparatively faintly with any aniline dye, but (like the micrococci) were Gram-positive.

Culture media inoculated with the contents of the pipettes only developed pure colonies of the coccus. Even ordinary broth-tubes did not show any development of the short bacillus, which was evidently an an anaerobe, as it ultimately proved to be. The coccus in its appearance, mode of growth on media, &c., proved to be identical with that found in previous outbreaks of gangrenous mammitis in sheep, and with that described by Nocard.

Experiments.

In order to ascertain if the presence of the bacillus had any effect, two ewes of the same breed, each suckling a lamb, were chosen for experiment. One (No. 73) was inoculated with about $\frac{1}{10}$ c.c. of material from a pipette, the other (No. 74) with the same quantity of a pure broth culture of the diplococcus, in each case the material being mixed

with ten times the quantity of sterile broth, and carefully injected into the galactopherous sinus of the right half of the udder without injury to the mucous membrane.

The results were as follows: The right udder of No. 74 in twenty-hours was much swollen, hot, and painful; the secretion was curdly, and showed many cells with numerous cocci. In forty hours the gland was discoloured, but not intensely; swelling of the skin in front of the udder was observed; the animal was dull, moved very stiffly, and rumination had ceased. On the third day the swelling had increased, and was displacing the left udder, which, however, remained normal but for the appearance of many lymphocytes in the milk. The milk-secretion was deeply blood tinged, and very sparse in amount. The subcutaneous abdominal swelling extended forward for about 10 in., and was tense and hand. The animal walked with difficulty, and could not rise from the recumbent position without assistance. On the fourth day the udder was distinctly dark-violet in colour, the skin being necrosed and abraded. The abdominal swelling had spread forward to the axillæ, with discoloration of the skin. The udder secretion was deeply blood-tinged, and showed many degenerated cell-elements and groups of cocci. The animal exhibited great prostration. The temperature throughout fluctuated slightly, but had remained fairly constantly above 104° F. On the fifth day the animal, being *in extremis*, was slaughtered.

Post-mortem Examination.

In ewe No. 74, the subcutaneous tissue around the whole udder and extending forward to the elbow was infiltrated with much greenish-tinted but otherwise clear gelatinous exudate. The right udder was gangrenous towards the exterior, and especially the posterior half, the lobules having almost completely disappeared. The anterior part showed many hæmorrhagic areas, the tissues deeply inflamed, and infiltrated with greenish exudate, giving it a peculiar marbled appearance. The left udder was practically normal, but for the subcutaneous effusion.

Considering the small quantity of culture injected, the course of the disease was fairly rapid.

Ewe No. 73, inoculated with material containing many diplococci and bacilli, developed no marked evidence of mastitis. Three days after inoculation the udder was to all appearances normal, but careful examination detected a distinct hardness of the teat-duct. The milk-secretion was small and somewhat adhesive. Smears showed num-

erous lymphocytes and polynuclear leucocytes, with many bacilli and diplococci, numbers of each being englobed by phagocytes. This condition persisted until ten days after inoculation, when the animal was subjected to another experiment.

It was evident that this ewe (No. 73) either possessed some degree of immunity to the coccus, or that the presence and development of the bacillus inhibited in some way the growth or virulence of the coccus. Provided the latter were the correct position, it might be that some artificial immunity to the coccus in a state of purity had been acquired. To test this 0·1 c.c. broth culture of the coccus (subculture from sheep No. 74) was injected into the *left* and normal galactopherous sinus. The result was increase of temperature in twenty-four hours to 105·8° F., slight swelling in both udders, with increase of milk-flow. Forty-eight hours after inoculation the animal was prostrate, the whole udder was swollen, and violet in colour, while the skin of both thighs was also deeply congested. The secretion from the *right* side was now for the first time markedly altered, it being dirty-brownish in colour, and on microscopical examination showed masses of the short bacilli, with comparatively few cocci. The secretion from the left udder was also slightly brownish-red and curdled, and smears showed many pus-cells and masses of cocci in a state of apparent purity. Death occurred about sixty hours after the second inoculation—*i.e.*, of the pure culture into the left udder.

Post-mortem Appearances.

The skin of the udder, thighs, perineum, and in front of udder was of a deep livid, almost black tint. Both sides of the udder on section were dark and necrotic, the only difference being the presence of more subcutaneous and general œdema in the left than in the right. The spleen was enlarged and pulpy; liver and lungs ecchymosed; the left ventricle exhibited some subendocardial hæmorrhages and the presence of an *ante-mortem* clot, in which several groups of cocci were demonstrated. Microscopical examination of material from the udder showed, as before, some cocci, but chiefly bacilli in the right half, and only masses of cocci in the left.

In this case the second inoculation had first stimulated the general secretion, and then established acute inflammation in both udders simultaneously, and so probably overcame the inhibitory action of the bacilli, even though they also increased rapidly in numbers.

Further Experiments.

The bacillus in question was isolated and cultivated in broth anaerobically, it proving to be (as in milk) non-motile, staining with Gram, and forming but a slight turbidity in the media. Ewe No. 76 was inoculated with 0·01 c.c. of this culture, mixed with an equal quantity of coccus culture. The udder remained practically normal, but for a slight hardening of the teat duct and the appearance of leucocytes in the milk for a few days, after which no abnormality could be detected.

As a control ewe No. 75 was inoculated with the same quantity of a pure subculture of the coccus. The result in this case was a comparatively chronic purulent mastitis, the udder becoming slowly harder, lividity only appearing at the end of a week, after which the udder contracted in size, the secretion became serous, finally ceasing. This reaction was not typical, and was probably due to the fact that the lamb had been weaned for a week prior to the inoculation, and that lactation had almost ceased. Both ewe No. 75 and ewe No. 76 were, however, in the same stage of lactation—that is to say, they were almost "dry." No lactating ewes were available for further experiments.

The evidence points, therefore, strongly in the direction of the bacillus acting in a manner inhibitory to the coccus, and explains the atypical nature of the symptoms observed by Mr. Reakes in the naturally affected cases.

Effect of the Coccus on other Animals.

Rabbit No. 223 was inoculated with ½ c.c. broth culture subcutaneously. Death occurred in twenty hours. *Post-mortem* examination showed only slight blood extravasation under the skin of the thigh, but otherwise no abnormality. The coccus could only be isolated from the region of the inoculation.

Rabbit No. 224 was inoculated with a similar dose. In twenty hours the animal appeared prostrate, the ears were cold, there was œdema of the scrotum and the thigh, and profuse urination was marked. At twenty-eight hours the animal, being *in extremis*, was killed for examination. *Post mortem* showed no abnormality, except the slight œdema already noted and a distended bladder. Smears from the muscles of the thigh showed many cocci, but in the blood and elsewhere none could be detected, and they proved absent even in cultures.

Guinea-pigs Nos. 849 and 850, inoculated with ½ c.c. broth culture:

In twenty-four hours a large painful swelling developed, and this next day had extended to the scrotum. This gradually decreased, ultimately becoming localized as a walnut-sized, dense tumour, containing pus.

Lamb No. 82, inoculated in thigh with ½ c.c. broth culture: In twenty-four hours a painful swelling was present, and the animal was distinctly lame; temperature, 103·5° F. The swelling rapidly increased, and by the following day the œdema had infiltrated practically the whole of the subcutaneous tissue of the leg downward to the hoof; temperature, 105·8° F. The skin rapidly became discoloured, and by the fourth day necrotic patches appeared. The temperature on the third day was 106° F., and on the fourth had dropped to 105° F. Lameness had been intense throughout, and by the fifth day the limb was completely paralysed, being dragged by the animal when forced to move. Emaciation had been rapid, and food was refused. On the seventh day the lamb, being in a pitiable state, was killed. There was no pus at the region of inoculation, but below the necrosed skin, and extending down the thigh and covering the muscles, was a thick layer of adhesive exudate. The subcutaneous tissue below the hock was very œdematous. The muscles of the limb were pale and degenerated. Cocci were found in the adhesive exudate in the muscles and the subcutaneous œdema, but the blood proved sterile.

Lamb No. 83, inoculated with ½ c.c. broth culture: This animal was observed distinctly lame four hours after inoculation. In twenty hours the whole of the inner surface of the limb was œdematous from the groin to the toe; temperature, 104·6° F. The following day the animal was very weak, could only rise when assisted; both groins were swollen and very congested; the temperature remained the same. Death occurred forty-eight hours after inoculation.

Post Mortem.—Subcutaneous tissues of inoculated limb infiltrated with a large quantity of blood-tinged serous exudate; peritoneum deeply congested; kidneys congested and enlarged, each weighing 4 oz. There were numerous subcapsular hæmorrhages, and the capillaries of the cortex and medulla were intensely congested. The mucous membrane of the abomasum showed acute congestion, with submucous hæmorrhages, and the intestines were congested throughout. There were slight hæmorrhages under the capsule of the spleen. The liver was acutely congested. In the right ventricle of the heart subendocardial hæmorrhages were marked. All the lymph glands were markedly hyperæmic. Smears from the subcutaneous œdema alone showed cocci, and media inoculated proved this organism to be

present in a state of purity. No cocci could be isolated from the internal organs or the blood.

The lambs were about three months old.

These experiments demonstrate that the virulence of this coccus for all animals inoculated was much greater than is ordinarily exhibited by the microbe of gangrenous mammitis, as previously isolated here and elsewhere, provided it was employed in pure culture.

No experiments with cultures of the coccus and of the bacillus mixed together were conducted on animals other than sheep as described, chiefly owing to the fact that the investigation was conducted just as I was leaving the Department; in fact, the last observations were made by me after I had actually left the service. The whole circumstances seem, however, to be worth recording, and to indicate that some interesting results might be gained by pursuing investigations with mixed cultures in this and in other diseases.

DISEASES OF CANARIES.

Indexed B. A. C

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From Christchurch last summer a peculiar disease affecting certain aviaries in the city was reported by a noted breeder of prize birds, who forwarded the body of one for examination.

History.

The history supplied was meagre. For some days an affected bird would appear drowsy, disinclined to eat, and preferring to rest on the bottom of the cage rather than perch. There was no diarrhœa or other definite symptom exhibited. Death generally occurred within a few days of illness first being observed, but frequently no symptoms were noticed, and occasionally again the animal would linger for much longer than the average period.

Post-mortem Appearances.

Post-mortem examination of the bird received disclosed no pathological condition further than a very enlarged and pulpy spleen, which measured an inch in length.

Microscopical examination of the blood and the spleen demonstrated some long sporulating bacilli and a number of short bacilli staining bipolarly. These were isolated: the former proved to be, as anticipated, putrefactive, and the latter was found to be pathogenic

for rabbits, guinea-pigs, mice, and pigeons, but not for ordinary fowls. Later on a cage was received from Christchurch containing three canaries; this was one of the cages in which a considerable mortality of canaries had occurred. It had been cleaned out, but probably not thoroughly disinfected. The three canaries sent in the cage had not been in contact with any diseased birds, nor in any premises where there had been a noticeable mortality. One was observed rather drowsy on arrival, but no other symptoms were exhibited. Next day the dulness had increased, while the respirations were slightly accelerated. This condition continued for twenty days after arrival, when the bird was found dead one morning in the cage. *Post mortem* exhibited nothing abnormal. There were a few bacilli in the blood, these being apparently similar to those previously isolated. That the bacillus was the same was proved by subsequent experiments.

Characteristics of Bacillus.

A facultative anaerobe, very irregular in length, varying from almost a coccus to the length of *Bacillus typhosus*, often in pairs, motile, staining bipolarly, and Gram-negative.

On agar the colonies are large, circular, with smooth edges and raised nipple-like centre, greyish-white and moist-looking. In streaks the growth is broad, raised in the middle, with fairly regular edges, which tend, however, to send off shoots.

On gelatine at 22° C. it grows readily, the appearance being similar to that on agar, but the edges of the growth are smooth. There is no liquefaction.

Broth (neutral) becomes rapidly cloudy, but no scum and little deposit is observed, the cloudiness being very uniform.

On serum the growth is not marked, and is not special. Milk is not curdled, although the bacillus grows readily in this medium, but being quite spherical in shape. Neutral-red is completely decolorized, in shake agars within forty-eight hours and in broth within twenty-four hours, the surface of the neutral-red broth showing a greenish-yellow tinge.

In glucose, saccharose, dulcite, and mannite media the growth is luxuriant. In the broth there is a very dense growth, much more opaque than an ordinary peptone broth. In the agar, as in shake cultures, there is evolution of gas, and a distinct alkaline reaction, especially with saccharose.

On potato there is a thin, almost invisible growth, which can only be demonstrated by scraping with the needle.

Indol is not formed. It will be noted, therefore, that the bacillus does not belong to the coli group.

Experiments on Animals.

Fowls.—Two were inoculated, one being an old rooster, with 1 c.c. of broth; the other, a young, but adult fowl, with 0·5 c.c. Both remained normal, showing no swelling or evidence of discomfort.

Pigeons.—Five were inoculated with doses of broth culture varying from 0·25 to 0·5 c.c. Two which received 0·25 c.c. died within twenty-four hours; one which received 0·25 c.c. died in forty-eight hours; one receiving 0·5 c.c. died in forty-eight hours; and one receiving 0·25 c.c. in three days. *Post mortem* showed little swelling at the seat of inoculation beyond necrosis of the adjacent muscles, which were pale and friable. The internal organs were normal, but for the heart, which in the majority of cases showed blood extravasation of considerable extent around the auricles. Microscopical examination of material from the region of inoculation showed enormous numbers of bacilli, but the blood contained very few, frequently only one or two being visible in one field of the microscope.

Rabbits inoculated with broth cultures in doses of 0·5 c.c. and under succumb in from two to five days. There is little or no œdema at the seat of inoculation, but occasionally slight hæmorrhage subcutaneously. The muscles at the seat of inoculation are swollen and very pale. The internal organs are normal in appearance, the spleen sometimes only being slightly enlarged.

Guinea-pigs inoculated with 0·5 c.c. broth culture die in four to five days, the *post-mortem* appearances being similar to those of rabbits, with sometimes a caseous adhesive deposit underneath the skin at seat of inoculation.

Mice inoculated with one drop of broth culture succumb in four to five days, with little pathological change beyond slight œdema at the seat of inoculation, a small quantity of peritoneal effusion, and an enlarged and pulpy condition of the spleen. Microscopic examination shows many bacilli at the seat of inoculation and in the peritoneal fluid, but extremely few in the blood, spleen, and elsewhere.

Canaries.—One inoculated with one drop of broth culture exhibited no symptoms beyond a slight drowsiness during the last forty-eight hours of life, and was found dead on the morning of the fifth day. There was no swelling at the point of inoculation, but the adjacent muscles were pale and necrosed. Beyond a slight enlargement of the spleen, all internal organs were normal. Examinations of smears showed

numerous bacilli at the seat of inoculation, but extremely few in the blood and spleen—so few that rarely more than one or two could be observed in one field of a blood film.

Another canary, a very old bird, was inoculated with a similar dose, but beyond a passing dulness from which recovery was rapid no effect was observed.

Unfortunately, no further information could be obtained regarding the epidemic amongst the canaries, as those we secured were only sent about the end of the outbreak. There appears little doubt, however, about the contagious nature of the disease, and that the cause was the bacillus which proved so virulent for nearly all the animals inoculated, with the exception, curiously enough, of barnyard fowls.

THE NECESSITY FOR THE STANDARDIZATION AND STATE CONTROL OF VETERINARY SERA, AND BACTERIAL DIAGNOSTIC AND THERAPEUTICAL AGENTS.[1]

Indexed B. A. t.

By GEO. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.
Professor in the Royal Veterinary College, London.

Practically nothing has been done in the way of attempting to secure uniformity in the strength and preparation of the various biological products used for the diagnosis, cure, or prevention of infective diseases in animals. Consequently it is little to be wondered at that the use of those agents has not always been followed by the results that were expected, and that widely divergent results have been obtained by various practitioners. The object of this note is just to draw attention to the present state of affairs, and to suggest that it is advisable that steps should be taken by some competent authority to regulate the manufacture of those agents and to examine them from time to time to ensure that they are potent to produce the specific action of the respective agent.

The agents that come to one's mind principally in this connection are tuberculin, mallein, antitetanic serum, antistreptococcic serum, and blackleg vaccine. These, at any rate, are the chief bacterial preparations used by veterinary surgeons in the British Isles, and so, of course, appear to us as of the greatest importance. There are many manufacturers of these agents, and these manufacturers can place their products on the market without any guarantee of their efficacy.

In a congress such as this, where our chief concern is with matters relative to the public health, those agents necessarily claim special

[1] Read at the Congress of the Royal Institute of Public Health, Birkenhead.

attention that are connected with diseases transmissible to man, namely, tuberculin and mallein, agents used in the diagnosis of tuberculosis and glanders respectively. In any systematic attempt to eradicate these diseases, the diagnostic agents referred to will be, and are, called upon to play very important parts. Both diseases may be slow and insidious in their development, giving rise to little or no suspicion of their presence. In this condition they are frequently sources of real danger, and probably of even greater danger than the advanced and obviously diseased animals, because although the latter would no doubt be ejecting larger amounts of virulent material, their condition is readily observed and precautions can be taken against them, while in the other cases no danger is apprehended, and so no precautions against infection are taken. In fact, this is a common history obtained after human beings have become infected with that most fearful disease, glanders.

Hence it is that there should be no possible room to doubt the potency of the diagnostic agent, where failure may mean the death of an attendant from an incurable, and about the most loathsome, disease imaginable. And yet we are absolutely at the mercy of the manufacturer.

The failure of tuberculin may result in the distribution of tubercle bacilli in milk with perfect freedom. It may cause the retention of an affected animal in an otherwise perfectly healthy herd, and be a centre of infection for the rest. In such cases then, the assurance of the potency of the tuberculin is essential from the public health standpoint, from the farmer's standpoint, and from point of view of the veterinary surgeon using it, since his professional reputation may be at stake.

Failures in the efficacy of both mallein and tuberculin have been recorded, with a natural loss of confidence in their reliability as diagnostic agents. It is quite possible that those agriculturists who distrust tuberculin have some grounds for their distrust, not of properly prepared tuberculin, but of inferior products, or of old samples that may have lost their efficacy. One sometimes hears practitioners declare that they get more satisfactory results with one particular make of mallein, or of tuberculin, or of antitetanic serum, or blackleg vaccine. Such statements lack somewhat by their being rather indefinite, but they are begotten of practical experience.

Then, again, there should be some control over the indiscriminate sale of these products. Tuberculin, in particular, may lend itself to fraudulent use by dishonest dealers, some of whom have learned that repeated injections into a tuberculous animal may produce a temporary

immunity to its specific reaction. Hence the sale should be limited to veterinary surgeons.

I am not aware of any extensive or systematic examination of these agents as put on the market by different makers, with the exception of antitetanic serum. Various marketed samples of this agent, however, have been very carefully examined and standardized by Drs. Moller and Eichhorn, of the Bureau of Animal Industry of the United States of America, and they have discovered enormous variations. Taking the American unit as the standard of comparison, the immunizing dose for a horse being 1,500 American units, they found the necessary amount present in some manufacturers' products, but considerably less in others, while in one case there was actually less than one-third the required number of units to produce immunity.

This variation has been proved experimentally in connection with antitetanic serum, and I think we may safely take it as a fact that, in spite of the great difference in the method of preparation, a similar lack of uniformity in potency exists in many of the marketed samples of tuberculin and mallein, especially in the light of practical experience.

It is these variations then, and the disastrous effects that may result therefrom, that lead me to bring this matter forward, and to ask that some standard of uniformity should be fixed and enforced. It is not for me to suggest what standard shall be fixed in each case. A small committee of experts should be appointed to definitely fix a standard which should be zealously enforced. Moreover, they should determine the length of time the agents will with certainty retain their efficacy, and after which they should be withdrawn from the market as doubtful.

It seems to me that the authority that should take charge of the matter is the Board of Agriculture, as the agents referred to are veterinary in character; but, failing that authority, I would suggest the matter should be taken in hand by the authority whose duty it is to administer the "Food and Drugs Act," viz., the Local Government Board.

[After the paper had been read and discussed, the following resolution was proposed by Professor Wooldridge, and seconded by Mr. J. S. Lloyd, and was unanimously adopted: "In the opinion of the Section of Comparative Pathology and Veterinary Hygiene of this Congress, it is desirable in the interests of public health that standardization and state control should be instituted of the various biological products used for diagnostic and therapeutical purposes, such as tuberculin, mallein, antitetanic serum, and vaccines."]

Clinical Articles.

TWO CASES OF ACUTE LAMINITIS WITH FAVOURABLE TERMINATION UNDER ADRENALIN TREATMENT.

By C. H. HYLTON JOLLIFFE, F.R.C.V.S.
Veterinary Captain 1st Life Guards.

Case 1.—The subject was a Household Cavalry troop-horse (gelding), aged 4.

The horse was under observation at the infirmary on account of slight urticaria, and on August 3 last, whilst being exercised, led, round the barrack yard, he slipped and fell on the near side. He got up and was apparently none the worse. The following day there was slight lameness on the near hind. This lameness increased on August 5 and 6, and on August 7 the horse was exceedingly lame and putting little or no weight on the limb. He was therefore placed in slings on this date. The lameness was referable to the hock, which showed some general diffuse swelling, heat, and tenderness to pressure around the joint. On the morning of August 8 the horse was found cast and badly entangled in the slings, having apparently tried to jump out of them during the night. On being released he subsided to the ground and was found to be unable to rise. The horse was then seen by Mr. G. H. Williams, M.R.C.V.S., who had the slings again placed under him and endeavoured to haul him up. The horse made, however, no effort whatever to stand, and it was found necessary to let him down. He fed well during the day, and about 4 p.m. he got up by himself with ease. He was seen again by Mr. Williams that evening, who found the horse up, but quite unable to move—"tied to the ground"—and he regarded the condition as one of acute laminitis affecting all four feet, and ordered the shoes to be removed as soon as the horse lay down.

On the morning of August 9, I saw the horse with Mr. Williams. He was then lying on his side, having laid down about midnight and remained so. The pulse was slightly vuluminous, full, and frequent. Respiration accelerated; temperature 101·8° F. Further ineffectual attempts were made to get the horse up, and finally the slings were again placed under him. He was raised up with the chain and pulley-block into a standing position and his limbs placed vertically under him, but he resolutely declined to make the very slightest effort to stand, and hung limp in the slings, precisely as though he was com-

pletely paralyzed. He further gave no reaction to pin-pricks applied to the limbs, and the case had every appearance of one of complete general paralysis. There was, however, considerable heat in the feet and phalangeal regions, and very marked throbbing of the digital arteries all round.

The horse was now let down, and 1 c.c. of a 1 in 1,000 adrenalin solution was subcutaneously injected over the internal and external digital arteries at the level of the fetlock joint, both fore. He continued to feed well, in the prone position. About 4 p.m., he got up of his own accord, and when seen at 6 p.m. he was standing up, but practically immovable. His attitude was not the usual one of a horse affected with laminitis. He stood with all four feet well under him, and not particularly on the heels. He would frequently "paddle" with the hind feet. Aloes ʒvi. was administered.

August 10.—At 9.30 a.m. the horse was standing up. He was still more or less immovable, though less so than the previous evening and on the whole he appeared to show a slight degree of improvement. It appeared evident that the horse was affected with laminitis in the hind feet as well as in the front. It was therefore decided to inject adrenalin over the digital arteries of the hind limbs, but owing to the constant "paddling" and to the fact that the horse was incapable of standing on three legs with one held up, it was found impossible to carry out the injections with the horse in a standing position. It therefore became necessary to throw him, and whilst being "pushed" into position to adjust the hobbles he lay down of his own accord.

Adrenalin solution 2 c.c. of a 1 in 2,000 of normal saline was then injected on each side of all four limbs at the level of the fetlock joint (eight injections), and the horse was left lying down.

August 11.—The horse got up by himself early in the morning and could move more freely. He was purging, but fed well. Pulse and respiration showed improvement.

August 12.—Horse showed marked improvement. He could move comparatively easily, and when led out of his box he walked round the yard quite comfortably, though still evidently lame "all four." Pulse and respiration normal. Ordered ten minutes' walking exercise night and morning. Heat, pain, and swelling in the region of the near hock (referred to on August 7) had practically disappeared. Pulse and respiration normal.

August 16.—There was further improvement, and horse capable of trotting, though still lame all round. Increased local temperature

about the feet continued and there was no diminution in the throbbing of the digitals. There was some general œdema around the fetlocks. I ordered half an hour's walking exercise night and morning and wet swabs applied to the feet, and cold water bandages to the fetlocks and pasterns.

On August 19, there was further improvement and horse trotted quite easily, though he continued somewhat lame all round; 2 c.c. adrenalin solution 1 in 2,000 injected on each side of fetlock, all four (16 c.c.). Cold swabs, &c., and walking exercise were continued.

August 23.—Moved "short" all round and digital throbbing continued, though less pronounced. He was shod, and walking exercise increased to an hour night and morning.

August 28.—Except for moving slightly "short," the horse trotted practically sound.

September 19.—Horse was doing duty and action normal. Digital throbbing no longer apparent, and surface temperature of the feet normal.

The horse remains sound up to the time of writing (October 12).

Case 2.—The subject was a black gelding of the same class as Case 1. The horse marched from Pirbright to London, a distance of about 30 miles, on July 14 last, and that evening showed marked signs of exhaustion. Temperature slightly raised, pulse disturbed, respiration accelerated, complete anorexia, &c. The horse spent the whole of the following day lying down, and the next day (16th), on being got up, was found to be showing typical signs of moderately acute laminitis, both fore. Shoes were removed and poultices applied. A purgative was administered, and the horse was exercised for half an hour twice in the day and kept loose in the box. On the 17th, the severity of the attack had slightly increased, and 4 c.c. of a 1 in 2,000 adrenalin chloride solution in aq. dest. were injected on each side of the fetlock, both fore. There was no perceptible change in the horse's condition that day.

On July 18 his condition was unchanged. Walking exercise continued.

On July 19 slight improvement was noticed and maintained.

July 23.—Considerable improvement. There was scarcely any lameness perceptible walking, and the horse could trot easily.

July 25.—Only slightly lame trotting, and progressing favourably in every respect.

July 31.—No lameness. Discharged, recovered, for duty.

CURIOUS FOREIGN BODY IN A HORSE.

By GEORGE D. MARTIN, M.R.C.V.S.

Westcliff-on-Sea.

SOME little time ago a client of the writer, "thinking it might be of interest," as he said, brought to the surgery a glass tube with metal top, with the information that a horse which he had recently bought at a repository "had had a severe fit of coughing, during which he had coughed out this tube in one piece (it was brought to me in two pieces as at present) which broke on falling on the floor of the stable."

The tube in its whole state measured 10 in. in length and had a diameter of $\frac{7}{16}$ in.

I, of course, scouted the tale as an invention, but the client, a most reliable man and known to the writer for years, was quite hurt at this and averred that the facts were as above related. He, at the same time requested me to put in a tracheotomy tube for the horse which had delivered itself of the tube in question, which I did a few days later.

[Perhaps the horse in question, knowing his own infirmity and seeing the tube on the floor, had picked it up with the idea that he could so adjust it in his own trachea as to save his owner the veterinary surgeon's fee for tracheotomy! Really the intelligence of some roarers is wonderful!! This is only a suggestion, and our correspondence column will not be open for any discussion upon the point!—ED. *V.J.*]

TWO CASES OF CONGENITAL TAILLESSNESS (PEROKORMUS ACAUDATUS).

BY STAFF VETERINARY SURGEON SEIFFERT.

DURING last autumn manœuvres I had the opportunity of seeing a strong three-days-old calf which was born without a tail. It resembled the illustration of this peculiarity given in the surgery of Bayer and Frockner to the article by Dr. Th. Schmidt on "Diseases of the Tail." It also showed that there is truth in Schmidt's statement that malformations of the tail and cranial-cavity often co-exist. The left eye was completely missing in this case; the left eyelids were represented by a small cleft from which thin tears trickled. Palpation of the eyelids showed lack of the bulbus.

The second case was that of a circus stallion completely devoid of any trace of a tail. The croup was simply gradually rounded off; there was neither raising nor dropping of it nor any abnormal deviation of the direction of the hair announcing the place where the tail ought to be.

The hair was regularly grown, flat, glistening and gradually merging into the hairless skin of the upper half of the opening of the rectum.

(*Zeitschrift für Veterinärkunde.*)

Canine Clinical Notes.

NOTE ON A USEFUL METHOD FOR THE REMOVAL OF TEMPORARY CANINE TEETH.

By GEORGE D. MARTIN, M.R.C.V.S.
Westcliff-on-Sea.

EVERY practitioner with much canine work is no doubt familiar with the difficulty so often experienced in removing the temporary canine teeth in young dogs, and especially in those cases where the permanent tusks have nearly, or completely, erupted alongside them. Having tried all sorts of forceps and lancing the gums more or less severely, in my efforts to *pull* these teeth without breaking them and with so frequently disappointing results, I have for a considerable time now quite discarded forceps for the purpose, and my method is now as follows:—

Having secured the dog on the table with the hobbles, according to Hobday's method, in the ventral position, and administered either a general or local anæsthetic (local as a rule suffices), holding the patient's head with the opposite side to that about to be operated on and supported on a thickly folded cloth, the gum is freely incised with one long incision, following as nearly as may be judged, the anterior border of the root of the tooth to be removed, and extending quite down to and through the periosteum in all its length; then an ordinary bradawl of best quality (steel and $\frac{1}{12}$ in. wide at its cutting edge) is inserted in the incision just clear of the neck of the tooth—*i.e.*, level with the anterior border of the tooth and in the groove between it and the permanent tooth—using one's first finger on the instrument as a guard in case of slipping, and with a slightly boring side-to-side movement of the wrist the end of the bradawl is forced perpendicularly between the temporary and permanent teeth to a depth corresponding with one's estimate of the thickness of the tooth to be removed. When this is arrived at, the hand is lowered gradually, and the end of the bradawl in the same way, steadily, with the same boring motion, forced up towards the root of the tooth; one feels the tooth gradually loosen, and, when sufficiently so, the hand is raised and the tooth carefully levered out of its position. All that is necessary then is to grasp it with a pair of dressing forceps, and a slight twist is sufficient to free it from any gum attachment and to complete the removal. Hæmorrhage is sometimes fairly free for a few seconds, but soon ceases, and I have not had a single case of trouble from that cause.

TUBERCULOSIS IN A SCOTCH TERRIER.

By J. F. CRAIG, M.A., M.RC.V.S.
Professor in the Royal Veterinary College, Dublin.

Subject.—Scotch terrier dog about three or four years old.

History.—About a fortnight or so previous to my examination it was noted that the abdomen of the dog began to increase in size, although the condition did not correspondingly improve.

Symptoms.—The condition of the dog was poor, the abdomen enormously swollen and tense, almost coming in contact with the ground. The mucous membranes were pallid, the temperature 101·8° F. The appetite and bowels were normal. On auscultation the heart-sounds seemed to be slightly muffled. The respirations were not markedly increased on raising the hind limbs. Palpation did not reveal any wave of fluid to indicate ascites. On manipulation a well-defined swelling could be made out, this swelling being in contact with the anterior portion of the abdominal floor. A laparotomy was advised and decided on. With the usual precautions and under general anæsthesia, the abdomen was opened along the floor from the xyphoid cartilage backwards. A large dark swelling at once presented itself, which was well defined and about as large as the human head. It was attached in front to the lower portion of the diaphragm and proved to be developed in connection with the liver. The attachment to the diaphragm was broken down, but in doing so an opening was made into the swelling, which proved to be an abscess, the contents of which escaped through the abdominal wound. The contents were clear, of a reddish colour and contained yellow flocules. The abscess then collapsed, the sides and posterior wall were removed. Further examination of the liver disclosed a number of large tubercles, and the case being one of tuberculosis, I had the animal destroyed by chloroform.

On *post-mortem* examination it was found that this large abscess had developed on the posterior surface of the right half of the liver, and its walls were yellowish-white in colour. Four smaller abscesses varying in size from a hazel-nut to an orange were present in the left lateral and middle lobes, and numerous small miliary tubercles were scattered through the liver tissue. The hepatic and mesenteric lymphatic glands were enlarged to about the size of walnuts and contained white caseopurulent centres. There was present also a tuberculous peritonitis. Numerous small granulations and tubercles ("grapes") were scattered over the great omentum, the mesentery, stomach, and intestines, and posterior surface of the diaphragm.

A small tubercle about the size of a pea was present in the cortex of the left kidney. The spleen was free from tubercles. In the thorax a little tuberculous pleurisy was noted, but chiefly near the foramen sinistrum over the diaphragm. The lungs were studded with miliary tubercles. Tubercles were also found in the left bronchial, two prepectoral, and one tracheal glands.

Tubercle bacilli were present in small numbers in films from the lesions.

Remarks.—The chief interest in this case arises from the large size of the tuberculous abscess in the liver and the symptoms shown resembling those of ascites. Tuberculosis is not uncommon in the dog, and one of the most common seats of the lesions is the liver; dropsy of the abdomen being a common sequel.

A "ZULU CURE" FOR DISTEMPER.

By GEORGE D. MARTIN, M.R.C.V.S.

Westcliff-on-Sea.

One day last spring I was consulted by a gentleman home on a visit from Zululand, regarding his dog, an Aberdeen terrier of about a year old, which he feared was developing distemper. An examination on my part caused me to form the same opinion. Then the owner said he expected I should laugh and pooh-pooh what he was about to say, just as every other veterinary surgeon he had said the same to had done. However, I promised to listen with an open mind, and he then unfolded his tale.

In short, it amounted to this: that the Zulus believe the whole trouble in distemper is due to a worm in the under surface of the tongue which renders the tongue stiff and immovable, and thus causes inability to lap and to feed on the part of the dog, and that all the other symptoms result from this. The Zulus, my informant went on to say, are very expert in the removal of this so-called "worm," and he had seen many done and always with satisfactory results. Finally, he requested me to operated on his dog, the patient in question. After some considerable argument, in the course of which I endeavoured to convince him that the so-called worm was no worm at all, but merely what we call the median fibrous raphe of the tongue, and that it could not possibly exercise the influence attributed to it, &c., &c., my client was still quite unconvinced, and finally the question resolved itself into this: Would I do the operation for him? If I would not, having seen so many done in Zululand, he would try and do it himself.

Thus adjured I consented, and having anæsthetized the animal in the dorsal position, an assistant drew the tongue well forward and after wiping it free of mucus, spread it out carefully on a clean, dry cloth. I was surprised at the ease with which the raphe was removable, and at the very trifling hæmorrhage, and still more so at the result of the operation. The dog immediately on reaching home, one and a half to two hours after operation, ate his food with good appetite and to cut a long story short, from that day to this has never "looked back." All symptoms of distemper disappeared within two or three days and the dog required no further treatment at my hands! Though I have had many cases of distemper since that one, I confess I have not again tried that particular remedy. The fact remains that in this case no other treatment was attempted, that the dog absolutely refused all food and nourishment in any form before the operation, and after the operation appeared to forget all about having been ill. I do not attempt to explain the matter; I simply give the facts and the story as told to me, thinking it sufficiently curious to be of interest to readers of the Journal.

CUTANEOUS FILARIASIS IN A DOG.[1]

By S. N. MITTER.

Graduate, Bengal Veterinary College.

(From the Raymond Research Laboratory, Calcutta.)

On September 29 of last year, a Rampur hound was admitted into this College Hospital for treatment with the following clinical symptoms:—

Emaciation was very marked, and the animal was so weak that it could scarcely walk; there was a large patch of small pustules and ulcers, each surrounded by a hæmorrhagic zone, on the neck, extending downwards from which sero-sanious and sometimes sero-purulent fluid was discharged; the same kind of eruption was found on the hind legs. The dog also suffered from dysentery. The temperature and appetite were normal.

On examining, under the microscope, a drop of the fluid obtained by puncturing one of the fresh pustules, a small filarial embryo was discovered. The examination of some other pustules had the same result. A considerable number of similar parasites was also observed in the blood of the aural vein. In fresh specimens the embryos appeared as long, slender, translucent, apparently structureless,

[1] Reprinted from the *Journal of Tropical Veterinary Science.*

snakelike bodies wriggling about in the fluid in which they were found; the head was more or less rounded and slightly tapered; and the body was cylindrical and tapered gradually down to the point of the tail. The method of Sir Patrick Manson failed to demonstrate a sheath. This method consists mainly in preparing ordinary wet specimens of blood and then cooling them on ice for about eight hours; after removal from the ice the temperature rises, and the worm revives, and can be seen in the diffused hæmoglobin to be trying to break through his sheath. Under $\frac{1}{12}$ in. objective a fine transverse marking was observed in some preparations, especially at the caudal extremity. The mouth of the embryo was conical and showed no cephalic armature, but sometimes a small spine was seen protruding. The number of parasites in the different fluids varied considerably; in the pustules, one generally, but occasionally two worms were found; in a small drop of blood as many as five to ten specimens were seen. The examination of the fæces gave negative results.

In order to make a study of stained preparations, thick smears were dried in the air, the hæmoglobin was washed out with distilled water, and the specimen stained with weak solution of fuchsine, methylene blue, or hæmotoxylin and eosin. The embryo was then seen to be composed of closely packed granules of protoplasm. The length of the embryo was approximately 460 microns.

The local treatment consisted in applying tar and oil (1 to 40). The administration of creosote, bismuth, ipecacuanha and camphor cured the dysentery. General and nervine tonics were given during convalescence, and the animal was discharged quite cured on January 31, 1910.

An attempt was made to convey the disease by inoculation to a healthy dog, but without any results. Possibly an intermediate host is required, as pointed out in the observations on the metamorphosis of *Filaria nocturna* of man by Manson and of *F. recondita* of the dog by Grassi.

In *La Clinica Veterinaria* of 1906 there appeared from the pen of Dr. Dell'Aqua, a paper on "Canine Filariasis," in which he claimed to have found embryos of *F. immitis* in the liquid obtained from a fistulous ulcer in a skin affection of the fore and hind limbs of a dog which had been under treatment for two years; similar parasites were also discovered by him in the blood of the auricular vein.

"Cutaneous filariasis" in the dog has been observed by Rivolta, Siedamgrotzky, Moller, and others. There appears to be some

confusion about the biology of these parasites or possibly the disease is caused by different worms. Rivolta considers them to be embryos of *F. medinensis*. Siedamgrotzky is inclined to believe that they are *Anguilluale* (strongyloids).

The line of treatment adopted by Rivolta was the application of mercurial ointment. Siedamgrotzky's dog was cured by general cleanliness. Dell'Aqua's treatment consisted in the local application of 1 per cent. sublimate solution. Calomel ointment, intramuscular and intravenous injections of bichlorate of quinine, produced negative results. The dog gradually recovered, and the embryos and the cutaneous lesions disappeared.

[In conjunction with this note it is interesting to read the translation on p. 687 of "verminous dermatitis," &c.—Ed. *V.J.*]

PAPILLARY EPITHELIOMA ON THE TONGUE OF A HORSE.

By Staff Veterinary Surgeon Dr. KETTNER.

In a horse belonging to the third squadron of the regiment of Westphalian Lancers the tongue had been frequently noticed hanging out from the right angle of the mouth during riding exercise. An examination of the mouth was undertaken in consequence and disclosed on the left side of the tongue at the level of the small ligament a flat tumour, as large as a pigeon's egg, which was united to the under surface by a firm stalk. The pedicle passing from the tumour at its upper surface joined the under surface of the mucous membrane of the tongue, and was continued into the tissue of the organ by a stem as thick as one's finger. The external surface of the tumour showed at its centre an irregularly-shaped portion of a greyish-red colour as big as a two-shilling piece; on the edges and turned-up surface of the tongue, the colour was yellowish-white to pure white. On the whole of the upper surface of the tumour there were punctiform hæmorrhages. The edges of the tumour were rough and uneven. The consistency of the neoplasm except at the coloured parts, was hard. On section the upper part was greyish-red to greyish-white, indistinctly wavy and stripy, the yellowish-white edges, on the contrary, resembled a divided citron enclosing greyish-white fields of yellowish-white stripes.—Weight 20 grm.

Treatment consisted in removal of the swelling by putting a ligature round the stalk, and cutting off of the remainder with the scissors—hæmorrhage was quite inconsiderable. The horse did not evince pain during the operation which was conducted without any restraint, the patient only moving his head.

Complete healing took place in ten days; there was no return of the complaint. No sticking out of the tongue occurred afterwards.

The tumour was examined at the laboratory of the Military Veterinary Academy, and pronounced to be a papillary epithelioma.

From a forensic point of view the case shows that hanging out of the tongue may be due to a tumour.

(*Zeitschrift fur Veterinärkunde.*)

Abstracts.

SOME CONSIDERATIONS ON THE PHENOMENA OF PARASITISM AMONGST PROTOZOA.[1]

By Professor E. A. MINCHIN, M.A., F.Z.S.

A living organism, whether plant or animal, which gains its livelihood at the expense of another organism is termed a parasite, and the organism at whose expense it lives is termed its host. The conception of parasitism, therefore, implies an antagonistic relationship between two correlated beings, parasite and host, each of which has, so to speak, its own point of view, the one attacking, the other defending. The phenomena of parasitism are of most widespread occurrence, and very varied in their nature. There is probably no species of non-parasitic organism which is not liable to the attacks of some parasite, and the same holds true even of a great number of parasites themselves, which are subject to be attacked in their turn by some other parasite.

The problems of parasitism are very complex, and are also very important, both from the more abstract point of view, as problems of natural history and bionomics, and also from the practical point of view, in their bearing upon questions of medical and veterinary science. In a brief space it is not possible to discuss all aspects and problems of parasitism, nor can such a discussion lay claim to any finality in the present state of our knowledge. I propose to lay before you a few facts of parasitism, restricting myself entirely to the field of the protozoa, and to consider the phenomena solely from the point of view of natural history; a method of treatment which, though it cannot claim to solve all difficulties, may serve nevertheless to furnish certain guiding principles, and to throw some light, if only a side-light, on the questions at issue.

Among protozoa, as is well known, many different modes of life occur. We are concerned here with those species which live constantly in association with some other organism of a distinct kind. Such a species may live either upon, or in, the organism with which it is associated; in the first case it is then termed *epizoic*, in the second *entozoic*. In all such cases of association, the species which are truly parasitic must be clearly distinguished from those which are not. Taking first the case of epizoic forms, it is found that in many cases they merely utilize the body of their host as a coign of vantage where they readily obtain their food, or as a convenient means of transport, especially when the epizoic form in question is of a sessile habit of life. Every naturalist is acquainted with the sea-anemones that live upon hermit-crabs, to the advantage probably of both animals, at all events to the detriment of neither. There are many similar cases amongst the protozoa. As every microscopist knows, the appendages of many crustacea, especially of cladocera, such as *Daphnia*, and copepoda, such as *Cyclops*, are often thickly beset with sessile vorticellids, or acinetans, which obtain a convenient lodging but provide

[1] Reprinted from the Journal of the Quekett Microscopical Club, by permission of the President (Professor Minchin) and Committee.

their own board. Other forms occur on the stems of hydroids, as for example *Acineta papillifera* on *Cordylophora lacustris* (Hickling Broad, Norfolk). The common *Hydra* often bears a beautiful infusorian which from its louse-like appearance has the name *Trichodina pediculus*, but which caters for itself and not at the expense of the *Hydra*. Amœbæ are to be found creeping on the exterior of calcareous sponges, nourishing themselves on diatoms and other organisms. Similar instances could be multiplied indefinitely of epizoic species which are merely commensals, and not parasites in any sense of the word.

On the other hand, epizoic forms may be parasites, and even dangerous parasites, nourishing themselves at the expense of the animal they infest, and sometimes inflicting much damage upon it. It can easily be understood that an epizoic form which at first lived harmlessly upon some animal, drawing its supplies of food from the surrounding medium, might acquire the habit ultimately of obtaining its nourishment from the living substratum upon which it has planted itself. Examples of truly parasitic epizoic forms are the flagellate *Costia necatrix* and the infusorian *Ichthyophthirius*, both of which live on the skin of fishes and injure the epidermis.

In like manner, there are many entozoic protozoa which inhabit the bodies, and more especially the intestines, of other animals, but which are not to be considered as true parasites; they simply feed on various substances to be found there, such as waste particles of food or fæcal matter, or on other organisms, such as bacteria—in short, on material which, from the point of view of the host, is superfluous or even noxious. Thus entozoic protozoa may be quite harmless, perhaps even useful as scavengers to their host; but such forms may also readily acquire truly parasitic habits and become a danger to their hosts. A good example of this is seen in the amœbæ found in the intestine of man. One species, *Amœba coli*, which occurs commonly in man in all countries, is believed to be a harmless entozoic form. In the Tropics, however, other species occur which find a more abundant source of nutrition in the living body which harbours them; they attack the epithelium and tissues of the intestine, devouring cells and blood-corpuscles and giving rise to a virulent form of dysentery; and they penetrate into the liver, where they destroy and corrode the tissues, producing most dangerous abscesses. It is often very difficult to draw the line between harmless and harmful entozoic protozoa, but all such organisms must be considered suspect, and it is the safest course from the practical point of view to hold them guilty until they have been proved innocent.

The entozoic protozoa which are truly parasitic are found inhabiting a variety of situations in the bodies of their hosts. In some cases the host is another species of protozoon, into the body of which the intruder penetrates, living either in its cytoplasm or its nucleus. Amœbæ are very subject to the attacks of intra-nuclear parasites, and the young stages of many acinetans are parasitic upon other infusoria. When the host is one of the metazoa, the invading organism may be, in like manner, intra-cellular or intra-nuclear in habitat; or it may penetrate into the tissues, living amongst and between the constituent cells; or, finally, it may be found in one of the internal cavities of the body, such as the digestive tract, body-cavity, blood or lymph-spaces, urinary organs, &c., either living free in the cavity or attached to

the lining epithelium. We may distinguish, in general, two ways in which these parasites may tax the resources of the host. Some of those living in the digestive tract may simply absorb the soluble products of digestion occurring there, thus diminishing the nutriment of the host by intercepting its food supply. Other parasites, in the digestive tract or elsewhere, ravage the very substance of the body of the host, either by devouring cells and tissues, as in the case of the dysenteric amœbæ already mentioned, or, and more usually, by absorbing the vital fluids and juices of the cells, tissues, or organs into which they penetrate, sapping in many cases the life-springs of the organism that harbours them.

As diverse as the modes of parasitism amongst protozoa are the effects they produce upon their hosts. Some forms of these parasites cause no perceptible disturbance in the well-being of the host; even when they destroy cells and portions of the tissues, the damage may be slight, and is quickly made good without appreciable harm being done to the host. From this condition of more or less perfect harmlessness there is a continuous gradation in the ascending scale of capacity for harmfulness possessed by protozoan parasites, culminating in species which bring about the death of their hosts with greater or less rapidity. Hence, parasitic protozoa are commonly characterized as pathogenic, that is to say disease-producing, and non-pathogenic; these two terms imply, however, a distinction which is purely relative, since a "disease" may be anything from a slight and transitory indisposition to a severe and fatal illness. Hence, in discussing these parasites it is better to group them as lethal, or deadly, and non-lethal, including under the latter term both harmless parasites and those that produce ailments from which the host recovers more or less easily. The distinction between lethal and non-lethal parasites is not, however, in all cases absolute, since a parasite which is ordinarily harmless or but slightly harmful may exhibit lethal powers under certain conditions, especially when the host is in an enfeebled state of vitality from other causes. For instance, it is common to see animals living in captivity succumb to the effects of parasites which are harmless to them when living a natural healthy life in their wild state. The weakened powers of resistance of the host enable the parasite to flourish in an abnormal manner, and give it a capacity for harmfulness which it does not possess naturally. Hard and fast distinctions between different classes of parasites, from the point of view of the effects on their hosts, cannot always be drawn.

The great diversity in the effects of Protozoan parasites is very remarkable, and furnishes a most important subject for consideration and investigation. It would seem, at first sight, as if the presence of even a single parasite must cause some bad effect in the body of the host, even if the disturbance be so slight as to produce no appreciable symptoms of ill-health in the organism as a whole. If so, then the greater the number of parasites in the body of the host, or the larger the parasite itself, the greater should be the derangement in the health of the host. Consequently, the greater the powers of multiplication within the body of the host possessed by any given parasite, the more dangerous it might be expected to be. To a certain extent this expectation is realized. Thus the gregarines of the suborder eugregarinæ, found commonly in the bodies of insects and

other arthropods, are amongst the most harmless of parasites, so far as can be judged, and in these forms endogenous multiplication, as it is called, that is to say, multiplication in the actively parasitic phase in the body of the host, does not take place. On the other hand, the parasites of the closely allied order coccidia, begin their cycle of development in the host by very active endogenous multiplication, overrunning some particular organ or tissue, and producing a sick and enfeebled condition which brings the host often to the verge of death, and sometimes even kills it. As a rule, however, animals attacked by coccidiosis, as the disease is termed, recover from the malady owing to the fact that when the host is brought very low, its condition appears to react on the parasite, with the result that the parasite ceases to multiply endogenously, and enters upon a different reproductive phase; it produces resistant stages destined to infest new hosts, in the form of cysts and spores, which are inactive and do not absorb nutriment, but pass out of the body of the host. In consequence the diseased animal loses its parasites, or the greater number of them, and the tissues are left in peace to regenerate and repair their injuries, provided they have not gone beyond the point at which recuperation is possible.

If the action and reaction of host and parasite were relations dependent simply on the number or bulk of parasites present in the body of a given host, the problems of parasitism would be comparatively simple. Examples can be brought forward to show that in many cases the effects produced by protozoan parasites cannot be explained, either by the number of parasites present or by the aggregate bulk of the parasites in proportion to that of the body of the host. The effect produced by a given species of parasite on a given species of host is a specific reaction, which differs markedly when one of the two factors is varied. It is not uncommon to find insects with their digestive tract or body cavity crammed with parasitic gregarines, of relatively large size, but causing no apparent inconvenience to the host. On the other hand, mammals may be be killed by minute organisms in scanty numbers. A better comparison is furnished by considering closely allied parasites and hosts respectively. A rat may have its blood swarming with *Trypanosoma lewisi*, without being apparently any the worse for it. On the other other hand, in a man dying of sleeping sickness, caused by *T. gambiense*, or in a ruminant dying of nagana (tsetse-fly disease), caused by *T. brucei*, the trypanosomes may be so scanty as to be exceedingly difficult to detect.[1] These facts strongly suggest, as has been pointed out by medical authorities, that the parasites produce a specific toxin. Only in a single case, however, has it been claimed that a toxin has been isolated from a protozoan parasite, namely, the "sarcocystine," produced by the parasites of the genus *Sarcocystis* (sarcosporidia).[2] The subject is one which remains to be investigated, and in the present state of knowledge it would be unprofitable to discuss it further here.

The interaction of host and parasite is a question of great practical

[1] Compare Laveran and Mesnil, "Trypanosomes and Trypanosomiases," translated by Nabarro (Baillière, Tindall and Cox, 1907), pp. 146-150.

[2] *Comptes Rendus Soc. Biol. Paris*, li. (1899), pp. 311-14.

importance, which in consequence has been studied chiefly from the medical standpoint—that is to say, from the point of view of the host. I propose here, as I have already said, to deal with the question as a naturalist, and to discuss the problem rather from the point of view of the parasite; and with this object I shall begin by a brief consideration of the facts of parasitism in general.

A parasite is, like any other living being, an organism struggling for existence in a hard and cruel world. The difficulties, however, against which a parasite has to contend, in the struggle for existence, are different in many respects from those which beset a free-living organism. When once a parasite has obtained a footing in its proper host, the question of food supply is solved for it, since it finds itself lodged in the midst of abundant nutrition so long as its host lives. On the other hand, if the species is to be maintained, it is essential that the offspring of the parasite should be able to infect new hosts—a matter usually of great difficulty, and one in which the chances are all against the parasite in most cases. To ensure dissemination of the species a large number of offspring must be produced, and special adaptations and mechanisms may be necessary. Hence in parasites, the more they become specialized and adapted to parasitic life, the more the organs and functions of nutrition tend to become simplified, and the greater the tendency to elaboration of the reproductive system. It is especially amongst parasites that we find the greatest complication in the structure and mechanisms of the generative organs, the most extraordinary and delicately adjusted adaptations of the propagative phases, and the most astonishing fertility.

So long as the parasite has not made the necessary provision for propagating its kind and disseminating its progeny, it is against its best interests to kill or greatly weaken its host. The interests of the two are bound up together, and the death of the host, before the parasite has completed its reproductive arrangements, necessarily entails the extinction of the parasite. The ideal host, from the point of view of the parasite, is one that is "tolerant,"—that is to say, one that can support the presence of the parasite and keep it supplied with the nutriment it requires, like a good mother or nurse, without suffering in health and vigour to any marked extent. When once, however, the parasite has matured and produced its reproductive phases, the life or death of the host may be a matter of indifference to it. In some cases the death of the host may even be necessary for the dissemination of the parasite.

From these general considerations it is seen that reproduction, and, above all, the dissemination of the progeny in a manner adapted to the special circumstances of its mode of life, are by far the most important functions of a parasite. Turning now to the special consideration of protozoan parasites, we find that diverse conditions of their mode of life and habitat impose a corresponding diversity in their reproductive phases and their modes of dissemination.

The passage of a parasite from one host to another includes two manœuvres, so to speak: the passing out from the first host, and the passing into the second. Primitively it may be supposed that this migration was affected simply by the unaided efforts of the parasite itself—that is to say, that the active motile parasite would force its way out of one host, move freely in the surrounding medium, and sooner or

later attack and penetrate a second host. This primitive method of transference doubtless occurs in many cases, especially amongst epizoic forms. In the case of entozoic forms its occurrence is more doubtful. It is very probable, however, that the spirochætes found infesting certain bivalve molluscs, such as *Spirochæta balbianii* of the oyster, and *S. anodontæ* of the pond-mussel, swim out of one mollusc and into another. Active migration of this kind, however, is very rare amongst entozoic parasites, if it occurs at all. In the first place the conditions of life within a living body, in the midst of organic fluids, are so different from those in the open water, whether salt or fresh, that it is hardly to be expected that a delicate unicellular organism adapted to the one mode of life could stand the sudden change to the other. In the second place, it is clear that active migration of parasitic protozoa could only be effected when the host is an aquatic animal, and not when it is a terrestrial organism. The only instances of active migration of entozoic parasites known with certainty are those in which the parasite can penetrate a mucous membrane, and is thus able to pass from one host to another when two such surfaces are in contact. In this way the trypanosome of dourine in horses (*T. equiperdum*) passes from one host to another during coitus, and the transmission of the parasite of syphilis is another instance.

Speaking generally, and excluding for the moment those cases in which the transmission is brought about by means of an intermediary host, the propagative phases of protozoan parasites take the form of inactive, resting stages in which the body of the parasite is protected against adverse external conditions by a tough protective membrane. In the form of resistant cysts or spores the parasites in a dormant state brave the rigours of the external world, and are able to withstand the heat and drought of summer, the cold and frost of winter. Like seeds they offer an inert resistance to the elements and are disseminated passively, and like seeds they germinate when they reach a suitable soil, but not till then.

Let us consider now the manner in which the first step in the propagation is effected, that is to say, how the propagative phases leave the body of the host.

Many, perhaps the majority of protozoan parasites occupy positions in the body of the host whence the propagative phases can pass without difficulty to the exterior. This is the case when the parasite is lodged in organs which have ducts or passages opening directly or indirectly to the exterior; for instance, in the digestive tract and its dependences, such as the liver, salivary glands, malpighian tubules of insects, &c.; or the urinary organs and ducts. In all such cases the propagative phases of the parasite pass harmlessly to the exterior. The host may in this manner get rid entirely of its parasites, without however necessarily acquiring immunity to fresh infections; or, on the other hand, the parasite may keep up its numbers in the host by "endogenous" multiplication while at the same time it is continually sending forth the propagative phases destined to infect new hosts. In the majority of protozoan parasites the relations to the host are of this type, and the parasites are neither lethal nor pathogenic to any great extent.

On the other hand, there are many instances in which protozoan parasites occupy a position in the body of the host whence escape by

anatomical channels is not possible, or if possible, not suitable. This is the case when the parasite inhabits some enclosed space in the body, such as the cœlome or general body-cavity, or the blood-system; or when it attacks deeply-seated cells or tissues of the body. In such cases there are at least six known methods whereby the parasite is disseminated or transferred to fresh hosts.

(1) The resistant stages of the parasite may be set free by the death and decay of its host. This appears to be the manner in which some of the tissue-infecting parasites of the order Myxosporidia, especially the family *Myxobolidæ*, are disseminated. These organisms are for the most part parasites of fishes, and are often very deadly in their effects.

(2) The parasite may cause tumours and ulcers, which suppurate and so set free the spores or cysts of the parasite. This again is an effect commonly produced by tissue-parasites such as the *Myxobolidæ*. In such cases the parasite is always pathogenic and frequently lethal to the host.

(3) The parasite remains in the host until the latter is eaten by some animal which preys upon it. The propagative phases of the parasite are able, however, to resist digestion by the animal that has devoured their former host, and pass unaltered through its intestine, to be finally cast out with the dejecta. This is almost certainly the method by which the common *Monocystis* of the earthworm infects its host. The parasite produces resistant spores in the worm; the worm is eaten by a bird, mole, frog, or some other animal, through the digestive tract of which the spores of the *Monocystis* pass unaltered; they are scattered abroad with the fæces, and may then be swallowed by another earthworm, in which they germinate and produce an infection.

(4) As in the last case, the host, together with its parasites, is devoured by some animal, in which, however, the parasite is not merely carried passively, but becomes again actively parasitic. Hence in this case there is an alteration of hosts, one of the two hosts becoming infected by devouring the other. This mode of infection, which is well known to be of frequent occurrence amongst certain parasitic worms, such as cestodes (tapeworms), is probably also frequent amongst protozoa; but at present only one case of it is known with certainty, that of the species of the genus *Aggregata*, parasites of crabs and of cephalopods, such as the cuttle fish and the octopus. In the cephalopod the parasite forms resistant spores which pass out with the fæces and may then be devoured by crabs; in the crab the spores germinate and give rise to a second form of the parasite which lives and multiplies in its new host. If, as frequently happens, the crab is devoured by a cephalopod, the parasite completes its life-cycle by becoming once more a parasite of the cephalopod.

(5) The protozoa parasitic in the blood of vertebrates are disseminated in all cases, apparently, by blood-sucking invertebrates, such as leeches, ticks, or insects, which take up the parasites by sucking the blood of an infected animal. Later on the parasite may be inoculated into a second vertebrate host by the bloodsucking invertebrate when it sucks blood at a later feed. In some cases the transference of the blood-parasite from one vertebrate host to another

may be effected in a purely mechanical manner by the invertebrate, but in most cases the invertebrate is a true host in which the parasite multiplies and goes through a cycle of development. Hence, in such cases also there is an alteration of hosts and a complicated life-cycle, which has been most completely studied in the case of the malarial parasites of man, transmitted by the agency of mosquitoes.

(6) In some cases the parasite may penetrate the ovary of its host, pass into the ova, and thus infect the embryo and the next generation of the host. Transmission of this kind is known in a certain number of cases: for instance, in the "pébrine" disease of silkworms caused by *Nosema bombycis*. It is never the sole method of transmission, but is always supplementary to other methods.

To turn now to the methods by which protozoan parasites penetrate into new hosts. We find four known methods, which can be summarized very briefly after what has been stated already. The commonest is the method of casual or contaminative infection, where the host infects itself by taking up the propagative phases of the parasite accidentally from its surroundings. Most usually infection takes place by way of the mouth, with the food; but it may be effected by the respiratory organs. Other modes of infection are the contagious, as in dourine already mentioned; the inoculative, as in malaria and other diseases caused by blood-parasites; and the so-called hereditary method, as in *N. bombycis* and other cases.

From the foregoing summary of the methods by which protozoan parasites are propagated and spread from one host to another, it is clearly seen that there are only very few cases in which it is of direct advantage to the parasite to cause the death of the host, and even then only when the propagative phases of the parasite are fully matured. In cases where the parasite is disseminated by its host being devoured by another animal, it is necessary for the propagation of the parasite that the host should die a violent death, but not through the agency of the parasite; on the contrary, the interests of the parasite are best served by the host remaining in good health until the fatal moment arrives, unless it be supposed that some weakening of the host renders it more liable to become the prey of its enemies. Blood-parasites, transmitted by the inoculative method, are not perceptibly benefited by the host's illness, and certainly not by its death. It is, however, very necessary for this method of dissemination that the required phases of the parasite should be sufficiently abundant in the blood to make sure of infecting the invertebrate that sucks the blood and transmits the parasite. To ensure this taking place it is essential that the parasite should multiply and swarm in the blood to an extent that may, in many cases, lead to pathogenic and even fatal results. Hence one interest of the parasite may, so to speak, clash with another, and the all-important object of dissemination cannot be attained without bringing about a state of affairs which puts an end to the host and to the parasite with it; a result which, though detrimental to the parasite, may not be seriously so, if the death of its host do not supervene until there has been time for the dissemination of the parasite to have taken place. No reasonable person, however, could expect any adaptation to be absolutely perfect.

The inevitable conclusion from a general consideration of the facts

of parasitism amongst protozoa is, therefore, that it is hardly ever to the advantage of the parasite to be pathogenic, and still less so to be lethal to its host. This conclusion is borne out by the facts, for when the known forms of protozoan parasites are reviewed and considered in their entirety, it is found that the vast majority of them are quite harmless to their hosts. Pathogenic and lethal parasites are the exception amongst the protozoa, and are greatly in the minority when compared with the harmless forms. The attention of investigators has been so much focussed on the disease-producing forms, on account of their practical importance, that they appear at first to overshadow the harmless forms which obtrude themselves less on the public attention, though far more numerous. Thus, if trypanosomes are mentioned, most people think at once of the deadly parasites of sleeping sickness or other diseases; and it come almost as shock to find that the fishes and frogs of our waters, and the wild birds and animals of this and other countries, are commonly infested with trypanosomes without being perceptibly the worse for these parasites. In fact, the disease-producing protozoa are to be considered as exceptional and aberrant forms; the effects they produce on their hosts are detrimental to their own interests, and they are instances of what has been termed by Metchnikoff a "disharmony" in nature. How is the existence of these lethal forms to be explained, and what is their significance? The best manner for a naturalist to attack this problem is to consider groups in which both harmful and harmless forms occur and are represented by closely allied species. Good instances of such cases are presented by the trypanosomes of mammals, of which two groups may be briefly described and discussed.

In rodents we find a group of trypanosomes of which *T. lewisi* may be taken as a type, and I shall therefore refer to them for short as the *lewisi* group. *T. lewisi* flourishes in the two common species of rats (*Mus decumanus* and *M. rattus*), but not in any other animal, not even in the mouse, which has its own peculiar species of trypanosome (*T. duttoni*). Similarly *T. cuniculi* is specific to the rabbit, and *T. rabinowitschi* to the hamster. All these four trypanosomes of rodents are, however, very similar in their appearance and morphological characters, and can scarcely be distinguished except by the physiological action and reaction between them and their hosts; there can be no doubt that they are closely allied species, differing only in that each is adapted constitutionally to parasitism upon a special host. These specific trypanosomes of rodents of the *lewisi* group are further quite harmless, under ordinary circumstances, to their hosts. When a rat becomes infected with *T. lewisi*, the trypanosomes at first multiply rapidly and swarm in the blood; then all multiplication ceases and the parasites gradually die out, after which the rat is immune to the parasite, and cannot be infected with it a second time.

Contrasting with the *lewisi* group is another which we may term the *brucei* group, the members of which are remarkable for their deadly powers, each species being the cause of some fatal disease. Such are *T. brucei*, the cause of nagana, or tsetse-fly disease in horses, cattle and dogs; *T. gambiense*, the cause of sleeping sickness in man; *T. evansi*, the cause of surra in horses: *T. equiperdum*, the cause of dourine in horses; and several other species. In this group also the parasites are practically indistinguishable from one another by appear-

ance or structure; the species can only be identified by their reactions, but this is much more difficult in the *brucei* group, since, unlike the *lewisi* group, the species are not limited to one particular type of host. Every member of the *brucei* group can flourish in a great many different hosts, though there are limits even to their powers of adaptability. Thus *T. brucei* can flourish in ruminants, dogs, rats, and many other mammals, but apparently not in man. *T. gambiense* can infect man, monkeys, dogs, rats,&c., but not ruminants. When a rat is infected by *T. brucei*, the infection runs a course quite different from that of *T. lewisi*. The parasites in the former case multiply indefinitely until the blood contains more trypanosomes than blood-corpuscles, and the multiplication of the parasite is only ended by the death of the host and with it of the parasites.

Comparing and contrasting these two groups of trypanosomes, it is impossible to avoid in the first place the conviction that in each group the species are closely allied, and descended from a common ancestor. The ancestor of the *lewisi* group had certain structural characters, distinguishing it from that of the *brucei* group, but each of these two ancestral species has given rise to a number of species which are practically indistinguishable by structural characters, and can only be differentiated by their physiological reactions. We have here a beautiful instance of species in the act of arising, and caught, so to speak, at a stage in which divergent physiological adaptation has not yet brought about structural differentiation. It is seen further that the species of the *lewisi* group are quite specific in their reactions, and are each limited to certain definite hosts, while those of the *brucei* group are far less specific, and are limited only to a comparatively wide range of hosts. We may reasonably regard the *lewisi* group as the further advanced in the evolution of specific characters, and the *brucei* group as being in a more incipient stage of specific differentiation. Finally, it is to be noted that the *lewisi* group are typical harmless parasites, while the *brucei* group are characteristically of the lethal type.

Further light is thrown on this question by a very interesting discovery made by Bruce with regard to *T. brucei*. He found that in districts where tsetse-fly disease was rife, *T. brucei* was to be found as a natural parasite of wild game, of which it is apparently as harmless as *T. lewisi* to rats. By the agency of tsetse-flies, *T. brucei* is taken up from its natural hosts, and inoculated into domesticated animals of various kinds, in which it flourishes, and to which it is extremely fatal. To the native races of domestic animals it is less deadly, but imported animals, new to the country, succumb rapidly. Thus the wild game acts as a "reservoir," from which the infection of *T. brucei* spreads to other animals.

From these facts it is seen that *T. brucei* is most harmless to those hosts in which it is a "natural" parasite, that is to say, in which there is what may be termed a racial adaptation of the hosts to the parasites, and which are constitutionally able to withstand the disease-producing powers of the parasite. On the other hand, it is most deadly to those races in which it is a new parasite, and which possesses no constitutional power of defence. The state of affairs is perfectly comparable to that seen in such a disease as measles, which is a comparatively harmless disease in Europeans, but an extremely

deadly disease amongst races in which it is freshly introduced. What is the precise nature of the mechanism of attack and defence is the fundamental problem of the researches on immunity which are now being conducted with such ardour and industry by pathologists all over the world, and which cannot be discussed now. The facts of parasitism indicate clearly, however, that when a race of hosts has been long associated with a race of parasites, the hosts gradually acquire constitutional powers of resistance to the parasite, which the hosts do not possess when first brought into relations with the parasite. Hence it may be said that a new parasite will generally be a dangerous one, though it does not necessarily follow that the converse is true, and that a dangerous parasite is always a new one.

Here it must be pointed out that there are two ways in which a given species of parasite is "new" to a species of host. On general grounds we must suppose that a parasitic organism of any kind is descended from ancestors which were not parasitic. Hence there must have been a period in the evolution of any parasite when its ancestors were first adapting themselves to parasitic life and establishing themselves in the body of their host, overcoming all resistance to their invasion. This kind of newness was probably not accompanied with lethal powers on the part of the parasite, since the capacity for reciprocal attack and defence on the part of the two organisms concerned was probably developed gradually in each organism and would tend to balance each other. The newness which is dangerous is seen where an organism with fully developed powers of parasitism in one "tolerant" host acquires the capacity for living in another host not adapted to. This consideration throws perhaps some light on the disease-producing powers of trypanosomes in general. From the known facts of the development of trypanosomes, it seems highly probable that they have in all cases two hosts, a vertebrate and invertebrate. There are further many considerations which indicate that they were originally parasites of the digestive tract of the invertebrate host. This view may explain why these flagellates are always perfectly harmless to the invertebrates, their "old" hosts, and only pathogenic to vertebrates, their "new" hosts.

Returning now to the consideration of the examples specially selected, it is seen that the harmlessness of the *lewisi* group is due to the fact that the species are specially adapted to certain hosts and cannot maintain themselves in any other but those particular hosts. On the other hand, the lethal powers of the *brucei* group are associated with the fact that its members, even when specially adapted to a "natural" host, can nevertheless establish themselves also in a wide range of "new" hosts, with fatal results to the latter. If I am right in regarding the *lewisi* group as further advanced in specific differentiation than the *brucei* group, it then becomes probable that the natural tendency of evolution is to bring about a balance in the profit and loss account of parasite and host, and that the species of pathogenic trypanosomes of the *brucei* group would tend in the future to become harmless parasites of certain hosts. The evolution, however, would be a long and gradual one, perhaps attained only by a painful process of natural selection of the hosts.

On the other hand, if I am right in regarding the members of the *brucei* group as newly-arisen species descended from a common

ancestral species of trypanosome, it follows that the ancestor must, through some process of variation, have developed the power of establishing itself in new hosts and invading, so to speak, fresh pastures. It may be supposed, therefore, that if this has happened once it may happen again, and that a parasite which is now specific to a single tolerant host may, by some unexplained process of variation, acquire at any time the power of infecting other hosts and so giving rise to new species of parasites ultimately. The facts of variation are at once the most patent and obvious characteristic of living beings, and the most difficult to trace to their causes; their study is of the utmost importance for understanding the evolution and the origin of species, and the phenomena of parasitism offer a field for the study of variation of a peculiar kind, that is to say, variation in physiological and constitutional than morphological characters. The variations in the powers of parasitic organisms are clearly of the greatest practical as well as theoretical importance, when it is seen that the spread of a parasite into new hosts is likely to be accompanied with the most deadly results to the hosts invaded by it. In fact, the origin of species amongst parasites is bound up with the question of the origin of disease. Ray Lankester has suggested[1] that the extinction of animals seen in past geological periods may have been in many cases due to their extirpation by some species of parasite new to them.

Thus it is seen that in the origin of species amongst parasites there are, as in other organisms, two steps: first the appearance of variations, with the resultant disharmony seen in the lethal forms; secondly, by a gradual process of reciprocal adaptation between host and parasite, the establishment of more normal harmonic relations, associated with definite specific characteristics and reactions on the part of the parasite and host.

OSTEOPOROSIS AFFECTING HORSES IN CEYLON.[2]

By G. W. STURGESS, M.R.C.V.S.

Government Veterinary Surgeon, Ceylon.

The disease is also commonly known as Big Head, a term that at once calls to mind one of its most prominent symptoms. It is a constitutional disease occurring in both young and adult horses, characterized by stiffness and lameness, with enlargement, softening, loss of weight from absorption of earthy salts, and fragility of the bones, more especially those of the upper and lower jaw (hence the term Big Head). Any part of the bony framework may be affected, and such a bone as the shoulder blade may be enormously swollen with minor head symptoms. The disease is recorded in Ceylon, India, Singapore, Burmah, China, Australia, Africa, America, and Sandwich Islands. There is little reference to it in English or Continental veterinary literature, which indicates that the disease is not common there, but odd cases have been recorded. The geological formation of a country does not appear to have much influence. It is very common in the volcanic Sandwich Islands, and everyone is painfully aware of its

[1] *Quarterly Review*, No. 399 (July, 1904), p. 134.

[2] From the *Tropical Agriculturist.*

prevalence in this country (Ceylon), which is not volcanic, but formed of ancient crystalline rocks. Limestone occurs in localities, but is generally deficient. I believe cases occur in every part of Ceylon. This may be due to the fact that most horses are purchased in Colombo and taken to all parts. They also move about the country in the course of work, sale or exchange.

It is a common belief that damp districts are favourable and dry districts unfavourable to its development. All the time pony-breeding was carried on at Delft, as far as my connection goes with it, I never met a case. This refers to a point with reference to grain foods which I shall come to later. The ponies received no food other than the grazing obtained on the island. The soil is mainly sand, with broken coral and sea-shells, and so contains a good deal of lime. Our knowledge as to the true cause is very deficient. Many eminent men think it is principally due to dietetic, and others to climatic, influences. Some regard it as a bacterial disease and infective. This is hardly the occasion upon which to examine the various theories in detail, but as regards the climatic and dietetic theories it may be pointed out that the disease is well known under very diverse conditions of climate and diet, as will at once occur to you from the list of countries in which it is common. The bacterial cause has some very firm believers, especially W. Robertson, of Cape Colony, who has attempted its investigation, and says he considers it undoubtedly contagious, notwithstanding the fact that he was not able to find any organism in the blood or diseased tissues, or successful in communicating it from one animal to another by inoculation or feeding upon diseased tissues. He states that the presence of a sick animal may infect a stable, and a healthy animal put into such a stable may contract the disease. Professor Law, of Cornell, states that, in the opinion of several city veterinary surgeons, a fresh horse put into the stall of one that had suffered from osteoporosis soon contracted the disease. I am much in the same position as Mr. Robertson. I have studied the disease for many years, but have not yet succeeded in finding any organism or communicating the disease to another horse by experiment. If it is an infective disease, how infection is carried is at present a mystery. In the absence of any knowledge as to the true cause it is better to keep an open mind.

I have now to mention what is to my mind a very important matter, and the views expressed are supported by much of my own experience. Some time last year a paper was published by Mr. H. Ingle, late chief chemist of the Transvaal Department of Agriculture, who analyzed healthy and osteoporotic bones and certain of the foodstuffs of South Africa for the purpose. It is not necessary here to give figures and analyses in detail. The conclusion he arrived at is that the abnormal condition of the bones is favoured by the use of food not necessarily deficient in lime and phosphates, but in which the ratio of the lime (and perhaps the magnesia) to the phosphoric acid is too low. This would be the case when the diet is composed of cereals only, which contain a large percentage of phosphoric acid and low percentage of lime. He does not discard the bacterial theory, and points out that such a diet, if not actually causing osteoporosis, may cause such a condition of the system as to favour greatly infection by an organism. His suggestions are:—

(1) To avoid an exclusive diet of cereals.
(2) To give grass or lucerne hay.
(3) Where No. 2 cannot be done to add lime as calcium carbonate or bone meal to the food.
(4) To segregate an affected animal. Under No. 2 such food as lucerne, clover, meadow hay, cabbages should be added to oats, oat straw, maize, barley and bran, any of which are bad as an exclusive diet, especially bran. Paddy and gram also come under this class. Mr. Bruce, of the Ceylon Mounted Rifles, kindly analyzed samples of paddy and gram for me, and found that both are deficient in lime and rich in phosphoric acid, instead of being about equal. Paddy 100 phosphoric acid to 15 lime, gram 100 phosphoric acid to 50 lime, and bran 100 phosphoric acid to 9 lime, oat hay (average South African) 100 to 51—as compared with English lucerne 100 to 478, meadow hay 100 to 262.

This theory deserves working out thoroughly by experiment. Most of us know the good effects of turning a horse out to grass. I know very well the stiffness occasionally met with in horses fed exclusively on oats, and in which I invariably find a very acid reaction in the urine. Change of diet and a little saline medicine soon puts matters right. The condition is not osteoporosis, but it may be akin to it. As accessory causes dirty, unwholesome stables, careless management, overwork, and exposure to wet and cold in early life, predisposes to the disease, and most cases occur under seven years, but old horses are sometimes affected. Shetland ponies have a bad name, and the worst cases I have ever seen in Ceylon were a pair of Shetland ponies which were only in the island a few months. The symptoms vary a deal. The common statement of an owner is that the horse has rheumatism. The usual symptoms are stiffness, lameness, in one or more limbs often fugitive, shortness of action, stumbling and general slackness. The appetite is often indifferent, with sometimes a dislike of corn and a relish for grass or green food of any kind. The abdomen becomes tucked up, giving a pinched appearance to the loins, which are often very weak. As time goes on the enlargement of the face is noticed—in the superior and inferior maxillary bones. The enlargement of the superior maxilla appears as a swelling on either side of the nose below the eyes, which become in my experience more almond shaped. The lower jaw may be enormously enlarged, so much so that it may hardly be possible to get the fingers into the cleft between the right and left bones. The emaciation, stiffness, and exhaustion advance until the horse may absolutely collapse, and fracture one or more limbs or the spine. In some instances, without any very marked symptoms, a leg may suddenly fracture when the animal is exercised or worked. I have seen both hind legs of a coach-horse fractured when starting to pull the coach, and sudden accidents on the racecourse are not rare. A horse suffering from the disease may be in the very best of condition. In some cases the disease seems to be arrested at a certain stage, and the horse may work for years, showing only more or less stiffness.

Professor Law states that excess of phosphates in the urine indicates an active disease process, and decrease of phosphates cessation of the process, giving some hope of recovery. The course of the disease may be very rapid, and end fatally in two or three months; in

other cases it is slower, six months or a year, even two years, and, as mentioned before, a horse may remain useful for several years. As regards treatment, when the disease is well advanced, treatment is, in my opinion, waste of money. In cases where it is suspected to be developing, my usual plan is to change the feeding, administer salines and limewater in the drinking water, and improve the hygienic conditions generally. I am informed that in Australia it often attacks young racehorses when first put into training; as soon as it is suspected, they are at once turned out to grass and recover.

What concerns us more is prevention, and here the best management and care will be attended with the best results. The stable should be bright, airy, and dry and clean, with good drainage; if dark, half a dozen glass tiles on the roof will improve matters wonderfully. Avoid overfatigue and exposure to wet, and, if the horse gets wet, have him well dried as soon as possible, and not left in the stable to dry. Avoid the frequent washing common in this country. I have had the best results from the regular administration of limewater in the drinking water, which is very soft in many parts of Ceylon. It must, however, be a regular practice, and not given for a week and forgotten. It is easily and cheaply made by adding a handful of unslaked lime to a bucket full of water—stir up—allow it to settle, then pour off the clear water for use and bottle it, and as a general practice I give half a pint in each bucket of drinking water. Carbonate of lime or sterilized bone meal may be given in the food in small quantities. If Mr. Ingle's theory is correct, and I am inclined to think there is much in it worthy of further experiments, it will be advantageous to import into Ceylon English lucerne, clover, and meadow hay, and use it with the usual Ceylon ration and get a chemist to draw up the correct proportions of a mixture to give a properly balanced ration. I have no doubt the importers of horse foods will do it if there is any demand, and the cost will not be very different from that of the foods now used. Some legislation directed against the disease has been urged upon me by horse-owners in this country for some time. In the absence of definite knowledge of the cause it is difficult to know what measures will be of service. In deference to the wishes of many owners the disease has been included in the New Diseases of Animals Ordinance for the first time in any country, I believe, and the measures proposed, if agreed to by Government, will be on the following lines:—

(1) Power will be taken to destroy without compensation any horse, ass, or mule imported showing definite signs of osteoporosis.

(2) Every person having in his possession or under his charge any diseased or suspected animal must report it.

(3) Any horse, ass, or mule declared infected shall not be kept or exposed in any public stable, and if an affected animal is kept for work a separate stable shall be provided, isolated from other stables.

(4) Advanced cases shall be destroyed and carcase disposed of in the manner directed.

(5) A stable in which a diseased animal has been kept must be thoroughly cleaned and disinfected in the manner to be provided. Special attention given to the floor, which, if earth, will be removed entirely, and cement or other floor to be disinfected as directed.

Review.

PIGS, PIGSTIES, AND PORK. By E. Mayall, M.R.C.V.S. Pp. 204, with 55 illustrations in the text. Published by Messrs. Baillière, Tindall and Cox, London. Price 5s.

Books on the pig, its treatment in health and disease, have not occupied much of the attention of veterinary authors, and the little brochure by Mr. Mayall is especially likely to find a place on the bookshelf of the agriculturist who desires to be "up to date." It is intended to supply useful hints to the farmer who desires to make pig-breeding a useful and profitable addition to his yearly income. For the veterinary practitioner whose lot is cast in a country district the book will prove interesting reading, but the author does not dive deeply into abstract questions of pathology, nor does he pretend to have written a deep treatise on the treatment of disease. That in itself would fill a volume. Simple treatments are given, but no prescriptions, and the chapter on disease is really of a "first aid" character.

The chapters on breeding and feeding are useful, as is also the one on the hygiene and construction of the styes. There are hints on the inspection of the carcase, and we hope that when the book runs into a second edition this chapter and the one on diseases will be elaborated, or, better still, perhaps the author may be induced to write a book for the veterinarian on these subjects alone.

Personal.

MR. W. S. MULVEY, F.R.C.V.S., has been elected President of the Central Veterinary Society for the year 1910-11.

MR. T. S. PRICE, M.R.C.V.S., the retiring President of the Central Veterinary Society, has been elected President of the National Veterinary Society for 1911, in the place of Mr. W. Woods, F.R.C.V.S., who has resigned. We understand that next year's meeting will probably be held at Carnarvon at a date in July close to that chosen for the Investiture of the Prince of Wales at Carnarvon.

MAJOR-GENERAL PRINGLE has been gazetted Director-General of the Army Veterinary Department in succession to Major-General F. Smith, whose term of office has expired.

PROFESSOR GOFTON'S paper, printed in our issue of October, was read at a meeting of the Association of Veterinary Officers of Health at Glasgow.

Translations.

AN INTERESTING CASE OF URTICARIA IN THE DOG.

By RAITSITS.

During a walk in the street a watch dog, aged 6 months, was affected with a severe œdematous tumefaction of the whole of the head dewlop and upper part of the neck. The tumefaction was badly defined, not very hot, not painful nor depressible. Besides the skin of the sides of the neck and anterior limbs showed rounded nodules from the size of a lentil to that of a haricot bean.

After administration of bitters and friction with alcohol the dog was entirely cured the day following.

The animal partook habitually of different kinds of vegetables; it was supposed that the eruption was due to the action of some vegetable substance.

The author had already observed a similar case in a fox terrier, the ingestion of strawberries or chocolate regularly produced an attack of urticaria.

(*Revue Générale de Médecine Vétérinaire.*)

DERMATITIS VERMINOSA IN THE DOG.

By Veterinary-Surgeon LIEBERT.

Hanover.

At the beginning of May of this year a brown, short-haired, one-year-old German pointer, apparently suffering from mange, was brought to the clinic for treatment. On the outer surface of all four extremities and the breast-walls up to half the height of the breast there were isolated hairless places as big as a mark piece and in other parts greater areas which were confluent and without well-defined edges. The skin was reddened here and covered partly with grey scabs and partly with nodules about the size of the point of a needle. Between these places there were individual scabs, and flat, lentil-sized pustules filled with yellowish-green purulent contents. There was slight itching. The form of the illness had a great similarity to scab. In a series of microscopic preparations made from these parts of the skin acari could not be established. On the contrary in every preparation a number of round worms were found-of slender form and with pointed tail-ends which moved about very vigorously in glycerine preparations. They had all about the same size, showed no outline of generative organs and no genital differences. They were evidently a youthful form of round worm whose kind could not be declared. The worms were situated chiefly in the scabs and pustule contents. But they were also present in those parts of the skin covered with scales although it was necessary to scrape the skin in order to obtain them; superficially-situated scales covered no round worms.

According to a previous experience in the clinic these worms

gained access to the dog from the straw, but although the owner was asked to send some of the straw used as litter for the dog he omitted to comply with the request and therefore a decision as to the species of nematode could not be given.

Since these worms of the dog disappeared without treatment it was resolved to adopt only expectant treatment in this case, the skin of the dog being frequently examined for the worms. As, however, a whole rank of worms was found after a week the skin was rubbed with 1 in 1,000 sublimate spirit. In the course of a few days the patient was free from worms. To help the growth of hair the skin was later on rubbed with peruvian balsam spirit. At the wish of the owner the patient was discharged after the above treatment. The bald places were not then quite covered with hair.

A similar case has been recorded by Künnemann in 1905:—

The worms in this case were recognized as a youthful form of *Rhabditis strongyloides*. They dwell in foul substances and cause the above described conditions when taking up their abode on the dog. They are not parasitic on the canine skin and do not depend thereon for a life-cycle.

Leunke in 1909 tells of some dogs being affected with these worms. Four subjects were attacked. The worms seen by him were half the size of muscle trichinæ.

Siedamgrotzki describes another case in which he found youthful worms and female nematodes in the straw used as litter, but no male examples. Besides these cases only two others have been described in veterinary literature by Rivotta and Schneider.

(*Deutsche Tierärztliche Wochenschrift.*)

[This case is especially interesting read in conjunction with the Clinical note by Mr. Mitter on p. 668.—Ed. *V.J.*]

HAVE BONY SWELLINGS AN INFECTIOUS CAUSE?

By Army Veterinary Surgeon MAGNIN.
Versailles.

Nobody will deny that large synovitic swellings frequently occur from an infectious cause, and perhaps the time is not far distant when many bony growths may be attributed to the same cause. German veterinary surgeons, and notably Eberlein, have been making investigations in this matter for some time, but hitherto exact proof of such an occurrence has been wanting.

The case recorded here by the author points very forcibly to the possibility of an infectious cause of exostosis. If a certain number of extensive swellings of the joints and tendon sheaths are caused by an acute infectious synovitis, why should not hard ossifying swellings arise also in the same way if the course of the complaint becomes more chronic?

A five-year-old horse at the remount depot fell ill from infectious pneumonia, which was very prevalent at the time. Four weeks later a painful synovitis showed itself suddenly on the right hind limb at the fetlock joint. It was of a subacute character, and caused great filling of the joint. A month later a similar synovitis attacked the left hind fetlock without any external cause being known; a short

time later the lameness disappeared under treatment, but three weeks afterwards it suddenly returned. Very soon after this there arose on the joint a rather voluminous, wide, bony growth, and almost at the same time such another exostosis appeared in the hock joint. Both spavin-like swellings caused great lameness, which was plainly shown to be advancing. The lameness also disappeared in a short time in this case.

The attack was interesting because the osteitic processes did not appear to be due in any way to bodily strain, and could only be ascribed to the earlier illness. The horse shortly afterwards became convalescent, and was gradually put to work. This case alone does not, of course, prove much, but a similar one has not been met with by the author in an extensive practice.

(*Deutsche Tierärtzliche Wochenschrift.*)

PROLAPSUS ANI IN A HORSE.

By R. W. GANNETT.

A WELL-NOURISHED brown gelding, with prolapse of the anus, had been treated by Gannett for ten days. The extruded portion was shown as a big round tumour, very susceptible to the touch and bleeding easily. With great straining hard, dry, blood-stained balls of dung were passed.

The rectum was carefully emptied, and aloes given as an evacuant. The next day the whole prolapse was ligatured in three portions, a part of the healthy mucosa being included in the ligature. For four days the dung had to be manually removed. The ligatured portion gradually sloughed away. Healing of the rectum quickly followed without further treatment.

(*American Veterinary Review.*)

AUTO-ENTERECTOMY IN A BITCH.

(From the records of the Laboratory of Veterinary Physiology in the Cornell University.)

FISH records the following unique case: A two-year-old collie bitch was spayed in the median line. On the evening of the second day after the operation it was noticed that the wound sutures were gone, and two free ends of intestine protruded from the wound. After irrigation of the abdominal cavity with adrenalin chloride and careful cleansing of the intestinal ends and removal of their gangrenous portions union was performed by Lembert's sutures (with catgut), and replacement effected. The skin wound was also sutured.

Shortly after the operation retching, finally ending in vomiting, occurred. The vomit contained blood-clots and two pieces of intestine, one about 30 cm. and the other 26 cm. in length. The blood-clots doubtless arose in the abdominal cavity when the intestine was bitten through, and by swallowing the two parts of intestine they had arrived in the stomach.

The bitch died naturally after the reposition. On section, clots

of blood were found in the abdominal cavity. How is it possible to explain the occurrence? Fish inclines to the view that a carnivorous instinct or a desire for cleanliness induces the animal to eat all flesh material derived from its body (*e.g.*, afterbirth). Flesh-eaters are, as a rule, very clean about their abodes; this desire for cleanliness will best explain why these portions of bowel were eaten. Noteworthy logic in which, perhaps, Mr. Fish alone believes.

(*Deutsche Tierärztliche Wochenschrift.*)

CONCERNING SOME AFFECTIONS HAVING THEIR SITUATION IN THE SINUS.

By Professor HENDRICKX.

Brussels.

After having recalled the classic notions of purulent sinusitis, the author indicates a systematic original treatment. He describes, besides two other congenital affections of foals:—

(1) *Purulent Sinusitis.*—Primitive (traumatic) or symptomatic of neighbouring lesions, dental caries, gingival shrinking, or rhinites. Diagnosed by unilaterality of symptoms: fœtid discharge, indurated adenitis of the nasal cavity, conjunctivitis. The deformation of the region, rare if the discharge is abundant, may be accompanied by dyspnœa and wheezing, produced from the side of the internal wall corresponding to the nasal occlusion.

Treatment by antiseptic fumigations and external medicaments is only a loss of time; early trepaning causes quick modification of the mucosa; the hardened pus is extirpated with a curette at its mossy edges, and a first lavage (never on the recumbent subject) of 10 to 15 litres of water at town pressure, or with a large syringe, is given by the only orifice of trepanation, the return of the water being hindered by a tampon, and thus for some days until disappearance of the clots and fœtor.

Then antiseptic injections, varied every five or six days, transform the purulent secretion into a mucous one. Finally astringent injections dry up the secretion, and we can let the orifice of trepanation close. Sometimes cure is completed by total filling up of the sinus, caused by settling of polypiform structures due to myxomatous hypertrophy of the mucosa. If dental caries is a cause it is necessary to remove the molar and continue the injections until the alveolus is obliterated, otherwise applying a tampon of gutta-percha conveniently softened.

(2) *Retention of Normal Mucous Secretion.*—This is hydropsy of the sinus, cited by Cadiot and Almy as due to partial or total obliteration of the cleft of communication with the nasal fossæ. It is evinced in the course of the first year by an insidious deformation a ways accompanied by nasal wheezing, without discharge or glandular enlargement) always visible enough on account of the deformability of the bone, whose synarthroses have not yet become solidified.

Trepanation gives issue to an aqueous liquid. The mucous surface often presents calcareous precipitates in plaques or stalactites.

Treatment consists in injections at high pressure, until some drops

of the liquid pass out at the nose; then two to three days after communication will be re-established, and in a few months the region will retake its normal form by natural force.

Four times the author has not been able to obtain the passage of a single drop of water by the nose. Three subjects have been slaughtered; on the fourth he perforated the partition of the inferior maxillary sinus, but this orifice was rapidly obliterated by cicatrisation.

(3) *Gaseous Accumulation in the Sinus.*—In foals of a month old, through congenital malformation of the cleft which acts as a valve; the cold air inspired dilates at the temperature of the body and distends the sinus, whose osseous walls have not yet solidified. Deformation is then slower than in the case of the retention cyst. The gas causing this tympanism is never fœtid.

(*Revue Générale de Médecine Vétérinaire.*)

PENIS PENDICULUS IN A PIG.

By Veterinary Surgeon GRABE.

Wittstock.

Gaabe records a case of penis pendiculus in a pig. The pig was a well-shaped store, whose penis was plainly visible hanging loosely between his hind legs, and possessed a bean-sized glans with an opened-out urethra. The prepuce was of normal size and in the middle of the belly. The pig was castrated as a youngster, and possessed normal testicles.

SADDLE PRESSURE.

By Chief Veterinary Surgeon Dr. BACKMUND.

Dr. Vogt expressed the view that the causes of saddle pressure should be sought in pathologic anatomical changes of the vertebræ, and in 1909 Hauptmann, of the Field Artillery, controverted the idea that "in saddle pressure the obstinacy of the horse must be overcome," and from his investigations asserted that the horse with saddle pressure on the vertebræ has or has had periostitis or ostitis, causing changes in the bone of the vertebræ.

As a contribution to the clinical study of the matter, I recently came into possession of a very instructive lumbar vertebra preparation.

The bones were those of an old grey mare of the riding-horse type which had been killed on account of lameness of the hindhand. On section, as a cause of the lameness, melanotic growths extending from the anus to the psoal muscles were found, which had converted the latter into deep black tumour masses. At the same time exostoses were found on the lumbar vertebræ.

When the specimen had been boiled, the preparation as illustrated, was encountered. The fourth and fifth lumbar vertebræ were united by a bridge of bone, as big as a pigeon's egg, extending from the left

to the middle line, and anchylosing them. The edges of the bodies of the vertebræ, although remaining free, were covered with little bony protuberances.

A further formation of bone was visible between the right transverse processes of the fifth and sixth lumbar vertebræ, whilst these were anchylosed by a flat exostosis of 3 cm. long. The joint on the left side showed no bony union, but some osteophyte formation.

In this case we have a spondylitis deformans. By periosteal extuberances bony growths arose which bridged over the space between the vertebræ and thus caused anchylosis. In men similar changes in the vertebræ have been described (see Kaufmann, "Text-book of Pathological Anatomy").

The commencement of such a process is considered in human pathology to take place in the bodies of the vertebræ from the lessened elasticity of the vertebral ligaments, and leads to the characteristic bone formation which manifests itself by alteration of the old spongiosa and a bony clasping of the intervertebral ligaments, the exostoses not being caused by pressure but by concussion which the vertebræ deal with.

This view of saddle pressure will gain ground according as cases are investigated, and it will be found that such subjects must be looked at more from the pathogenic anatomical standpoint, and given up to the veterinary surgeon rather than the dresser.

(*Deutsche Tierärztliche Wochenschrift.*)

Books and Periodicals, &c., Received.

Pigs, Pigsties, and Pork, by G. Mayall, M.R.C.V.S.; Mr. E. Merck's Annual Report of Recent Advances in Pharmacy and Therapeutics; Proceedings of the Royal Society of Medicine, Journal of the Royal Army Medical Corps; Rhodesia Agricultural Journal; Agricultural Journal of the Cape of Good Hope; Tropical Agriculturist; Bureau of Animal Industry, U.S.A.; Department of Agriculture, Victoria (Government Certification of Stallions).

Letters and Communications, &c.

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